RC71 B8
1884

Columbia University
in the City of New York

College of Physicians and Surgeons

Presented
Deposited by
Mrs. C. E. Gunther.

MEDICAL DIAGNOSIS

A MANUAL OF CLINICAL METHODS

BY

J. GRAHAM BROWN, M.D.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH; LATE SENIOR PRESIDENT OF THE ROYAL MEDICAL SOCIETY OF EDINBURGH

SECOND EDITION, ILLUSTRATED

"Felix, qui potuit rerum cognoscere causas."—VIRG., *Georg.* II. 490

BERMINGHAM & COMPANY

28 UNION SQUARE, EAST
NEW YORK

20 KING WILLIAM ST., STRAND
LONDON

1884

Medical Library

RC 71
B81
1884

PREFACE.

It is a creditable characteristic of the treatment of disease in the present day that it seeks to proceed on rational principles. Some there may still be who think it enough to give a name to a collective group of symptoms, and treat the disease as they have been told an affection bearing that name should be treated. There may be others who seize upon a few prominent symptoms, and direct their remedies exclusively to these. But every day is, happily, reducing the number of these routine practitioners, and teaching that the true physician is he who seeks thoroughly to investigate the phenomena of the disease, that in this way he may the better arrive at a knowledge of that from which they proceed, and to which, therefore, his treatment should be directed. But this can only be arrived at by a thorough knowledge of every change which disease produces in the body, and by a clear conception of what that change imports. This constitutes the science of Diagnosis, and, without accurate diagnosis, there can be no rational treatment.

The signs and symptoms of disease are changes produced in the animal economy, which are cognoscible by our senses—some by one, others by another; while to assist these senses we call in the aid of instruments which extend their range or increase their power, and of the various analytical processes which the science of Chemistry places at our disposal.

In the following pages an attempt has been made to describe these signs and symptoms of disease, and to show what is their value from a diagnostic point of view. If this attempt be at all successful, it may enable the student of disease to save much valuable time, by assisting him in analyzing and weighing the evidences of disease, and extracting from the whole phenomena which are presented to

him those which are of value as indicating its nature. The attempt is made not with a view of checking but rather of encouraging minute inquiry, while it aims at giving to the result of that inquiry more definite form.

A man who has clearly grasped a case in its entirety, who has separated the essential from the accidental, and who has ascertained the weight and bearing of each individual symptom, can go steadily forward in the treatment of his case without experiencing that harassing doubt which arises from partial or crude observation, and which, to a conscientious mind, cannot but prove a severe trial.

I desire to express my thanks to my friends who have encouraged and aided me in carrying out my design; among others, to Professor Grainger Stewart, from whom I have uniformly received much kind sympathy and advice. To Professor C. S. Roy I am indebted not only for the heart and pulse tracings with which I have illustrated Chapter XIII., but for very valuable assistance which I have received at his hands. Dr. Alexander R. Coldstream, of Edinburgh, has materially assisted me in the correction of the proof-sheets.

J. G. B.

63 Castle Street,
Edinburgh, 15*th September*, 1882.

TABLE OF CONTENTS.

PAGE

INTRODUCTION 13

CHAP. I. **The General Aspect, Condition, and Circumstances of a Patient** 15
- Family History 16
- Habits and General Surroundings 16
- Previous General Health 16
- Origin and Course of Illness 16
- Present Condition of the Patient 17
- Height and Weight 17
- Development and Muscularity 17
- Changes in the Color of the Skin 18
  - Pallor 18
  - Redness 18
  - Cyanosis 18
  - Jaundice 18
  - Bronzing 19
  - Grayness 20
- Œdema of the Skin 20
- Emphysema of the Skin 20
- Perspiration 20
- Expression of the Face 21
- Temperament, Constitution, or Diathesis 21
  - Sanguine Constitution 22
  - Nervous " 22
  - Strumous " 22
  - Lymphatic " 22
  - Bilious " 22
  - Gouty " 22
  - Rheumatic " 22
- Attitude of the Patient 23
- Evidence of Previous Disease 23
- Temperature 23

CHAP. II. **Alimentary System** 25
- Condition of the Lips 26
  - Color 26
  - Form 26

CHAP. II.—*Continued.* PAGE

Movements ... 27
Condition of the Teeth ... 28
Condition of the Gums and Mucous Membrane of the Cheeks ... 28
Condition of the Tongue ... 28
Form of the Tongue ... 28
Movements ... 29
Surface ... 29
Fur on the Tongue ... 30
Saliva ... 31
Fauces ... 32
Mastication ... 33
Deglutition ... 34
Examination of the Œsophagus ... 34
Appetite ... 36
Thirst ... 36
Sensations during fasting ... 36
Sensations after eating ... 36
Acidity ... 37
Flatulence ... 38
Nausea and Vomiting ... 38
Examination of vomited Matter ... 39
Defæcation ... 40
Character of the Fæces ... 41

CHAP. III. **Examination of the Abdomen** ... 42
Inspection of the Abdomen ... 42
General Prominence ... 43
Retraction ... 44
Local Tumefaction ... 44
Abdominal Movements ... 45

CHAP. IV. —— Palpation of the Abdomen ... 46
Condition of the Abdominal Walls ... 47
Condition of the Peritoneal Cavity ... 47
Condition of the Liver ... 48
Surface ... 48
Tenderness ... 48
Consistence ... 48
Size ... 49
Shape ... 49
Condition of the Spleen ... 49
" " Pancreas ... 50
" " Stomach and Intestines ... 50
" " Mesenteric Glands ... 51
" " Kidneys ... 52
" " Urinary Bladder ... 52
" " Abdominal Aorta ... 52

CHAP. V. —— Percussion of the Abdomen ... 53
Peritoneal Cavity ... 53
Liver ... 54
Spleen ... 57
Kidneys ... 58
Stomach ... 59
Auscultation of the Abdominal Organs ... 60

PAGE

CHAP. VI. **Hæmopoietic System**..... 60
Lymphatic Vessels..... 61
Inflammation..... 61
Narrowing and Dilatation..... 61
Rupture..... 62
Lymphatic Glands..... 62
Ductless Glands..... 63
Examination of the Blood..... 64
Microscopic Examination..... 64
Enumeration of the Blood-corpuscles..... 66
Estimation of Hæmoglobin..... 68
CHAP. VII. **Circulatory System**..... 69
Subjective Phenomena..... 69
Pain..... 70
Palpitation..... 70
Fainting..... 70
CHAP. VIII.——Inspection..... 71
Cardiac Impulse..... 71
Pulsation at the root of the Neck..... 72
Epigastric Pulsation..... 74
Arterial Pulsation on Thoracic Wall..... 75
CHAP. IX. —— Palpation..... 76
Cardiac Impulse..... 76
Alterations in Position..... 76
" " Strength..... 77
" " Extent..... 78
Double Apex-beat..... 78
Endocardial Thrills..... 78
Pericardial Friction..... 79
CHAP. X. —— Percussion..... 79
Position of the Heart..... 79
Absolute Cardiac Dulness..... 81
Relative Cardiac Dulness..... 81
Aortic Dulness..... 81
CHAP. XI.—— Auscultation..... 83
Heart Sounds in Health..... 84
Areas for Auscultation..... 84
Modifications of the Normal Sounds of the Heart..... 85
Variations in Intensity..... 85
Impurity of the Sounds..... 86
Reduplication of the Sounds..... 86
Murmurs..... 87
Endocardial Murmurs..... 87
Method of production of Murmurs. 88
Rhythm..... 89
Intensity and Propagation..... 89
Condition of the Normal Sound at the Orifice at which the Murmur originates..... 90
Mitral Murmurs..... 90
Tricuspid Murmurs..... 92
Aortic Murmurs..... 93

CHAP. XI.—*Continued.* PAGE

Pulmonary Murmurs..... 93
Murmurs of Non-Valvular Origin.. 94
Exocardial Murmurs..... 94
Pericardial Friction..... 94
CHAP. XII.—— Examination of the Arteries..... 95
Inspection..... 95
Palpation..... 95
Radial Pulse..... 95
Frequency..... 95
Rhythm..... 96
Character..... 97
Expansion..... 97
Tension..... 97
Volume..... 98
Percussion..... 98
Auscultation..... 98
In Health..... 98
In Disease..... 99
Cephalic Murmurs..... 99
Examination of the Capillaries..... 100
Examination of the Veins..... 100
Inspection..... 100
Auscultation..... 100
Bruit de Diable..... 101
Graphic Clinical Methods..... 103
Sphygmograph..... 104
Typical Healthy Curve..... 114
Anacrotic Pulse Curve..... 114
Dicrotic Pulse Curve..... 114
Hyperdicrotic Pulse Curve..... 115
Cardiograph..... 115
Sphygmomanometer..... 117
Note on the Measurement of Tracings.. 117
CHAP. XIII. **Respiratory System**..... 119
Subjective Phenomena..... 119
Cough..... 120
Sputa..... 121
Chemical Characters..... 122
Macroscopic Characters..... 122
Physical Characters..... 123
Microscopic Characters..... 124
CHAP. XIV.——Examination of Nares..... 128
Examination of the Larynx..... 129
Voice..... 129
Palpation of the Larynx..... 130
Laryngoscopic Examination..... 130
CHAP. XV.—— Inspection of the Thorax..... 135
Regions of the Thorax..... 135
Form of the Thorax..... 136
Respiratory Movements..... 138
Frequency..... 139
Rhythm..... 139
Cheyne-Stokes Respiration..... 139

CHAP. XV.—*Continued.* PAGE
Type 140
Pain and Difficulty in Breathing 140
Dyspnœa 140
Extent of Respiratory Movements 141
CHAP. XVI.—— Palpation of the Thorax 141
Vocal Fremitus 142
Pleural, Bronchial, and Cavernous Thrills .. 143
Fluctuation 143
CHAP. XVII.——Mensuration 143
Tape Measure 143
Callipers 144
Cyrtometer 144
Thoracometer 145
Stethograph 145
Spirometer 145
Pneumatometer 145
Theory of Percussion 146
CHAP. XVIII.——Percussion of the Chest 147
Methods of Percussion 152
Percussion Note of the Chest 152
The Intensity of the Percussion Sound 153
The Pitch of the Percussion Sound 155
The Tympanitic Percussion Note 156
Cracked-Pot Sound 159
Amphoric Resonance 160
The Feeling of Resistance during Percussion 160
Topographical Percussion 161
Regional Percussion 162
CHAP. XIX.—— Auscultation of the Lungs 163
Vesicular Breathing 164
Harsh Breathing 165
Jerky Breathing 165
Vesicular Breathing with Prolonged Expiration 165
Systolic Vesicular Breathing 166
Bronchial Breathing 166
Amphoric Breathing 168
Broncho-Vesicular Breathing 169
CHAP. XX. —— Adventitious Sounds accompanying Respiration 170
Moist Râles 170
Crepitant Râle 170
Fine and Course Bubbling Râles ... 171
Dry Râles 173
Sonorous and Sibilant Râles 173
Pleuritic Friction 173
Auscultation of the Voice (Vocal Fremitus). 174
Succussion 176
CHAP. XXI. **Integumentary System** 176
Subjective Symptoms 177
Eruptions 177
Distribution and Configuration 178
Elements of Skin Involved 178

CHAP. XXI.—*Continued.* PAGE

Type of the Eruption 178
Etiology of the Eruption 181
Vegetable Parasites 182
Achorion Schönleinii 182
Trichophyton 183
Microsporon Furfur 185
Animal Parasites 186
Sarcoptes Scabiei 186
Pediculus 187
Pulex Irritans 187
Demodex Folliculorum 187

CHAP. XXII. **Urinary System** 188
Subjective Symptoms 188
Examination of the Urine 189
Quantity 189
Color 190
Transparency 192
Odor 192
Specific Gravity 192
Reaction 194

CHAP. XXIII. —— Normal Constituents of Urine 195
Urea 195
Estimation by means of Nitrate of Mercury 196
Estimation by means of Hypobromite of Soda 197
Uric Acid 199
Creatinin 200
Indican 201
Cholorides 202
Sulphates 203
Phosphates 204

CHAP. XXIV.——Abnormal Constituents of Urine 206
Albumen 206
Paraglobulin 209
Propeton 209
Fibrin 210
Pepton 211
Mucus 212
Sugar 212
Blood 217
Bile Pigment 218
Bile Acids 218

CHAP. XXV.——Urinary Sediments 219
Blood Corpuscles 219
Pus Corpuscles 219
Epithelium 220
Renal Tube-casts 220
Spermatozoa 221
Micro-Organisms 221
Inorganic Sediments of Acid Urine 225
" " Alkaline Urine 225

CHAP. XXVI. **Reproductive System** 226

CHAP. XXVI.—*Continued.* PAGE
The Female Reproductive Organs and Functions 226
Menstruation 227
Amenorrhœa 227
Menorrhagia 227
Dysmenorrhœa 228
Leucorrhœa 228
Pareunia 228
Pregnancy 228
Physical Examination of Female Organs 228
Mammæ 228
Abdomen 229
External Pudenation 230
Vaginal Examination 232
Bimanual Examination 232
Speculum 233
Uterine Sound 233
Instruments for dilating the Cervix 233
CHAP. XXVII. **Nervous System** 233
Sensory Functions 234
Subjective Sensations 234
Pain 234
Paræsthesia 235
Giddiness 235
Abnormal Visceral Sensations 236
Cutaneous Sensibility 236
Common Sensibility 236
Tactile Sensibility 236
Sense of Pressure 237
Sense of Temperature 237
Sense of Locality 237
Muscular Sense 238
Special Senses 238
Sight 238
Diminution of Visual Acuteness 239
Alterations of Visual Field 239
Alterations in the Perception of Colors 240
Movements of the Eyeball 240
Paralysis of Ocular Nerves 240
Spasm of Ocular Nerves 241
Changes in the Pupil 242
Ophthalmoscopic Examination 243
Hearing 244
Taste 245
Smell 246
CHAP. XXVIII.—Motor Functions 247
Visceral Motor Functions 248
Voluntary Motor Functions 248
Voluntary Movements 248
Paralysis 248
Electrical Reactions 249
Spasm 250
Reflex Movements 251

CHAP. XXVIII.—*Continued.* PAGE

Superficial Reflexes..... 252
Deep Reflexes..... 252
Affections of Co-ordination..... 253
Labyrinthine Vertigo..... 253
Ataxia..... 253
Cerebellar Inco-ordination..... 254
Vaso-motor Functions..... 254
Cutaneous Vaso-motor Affections..... 254
Visceral Vaso-motor Affections..... 255
CHAP. XXIX.——Trophic Functions..... 255
Trophic Affections of Muscles..... 255
" " Bones and Joints.. 255
" " Skin..... 256
" " Secretory Glands.. 256
" " Viscera..... 256
CHAP. XXX.——Cerebral and Mental Functions..... 256
Coma..... 257
Illusions..... 257
Hallucinations..... 257
Delusions..... 257
Delirium..... 257
Speech..... 258
Sleep..... 259
CHAP. XXXI.——Condition of Cranium and Spine..... 260
CHAP. XXXII. **Locomotory System**..... 260
Condition of Bones..... 261
Condition of Joints..... 261
Condition of Muscles..... 261
Rigidity..... 261
Contracture..... 261
Flaccidity..... 261
Fibrillary Twitching..... 261
Appendix A.—On the Examination of the Fæces 262
Appendix B.—Method of Preparing the Solution of Nitrate of Mercury used for estimating Urea 263
Index..... 265

MEDICAL DIAGNOSIS.

INTRODUCTION.

A PHYSICIAN, when consulted by a patient, is naturally enough expected to be an attentive listener to what, to his informed mind, is a strange medley and most confused account of those deviations from health or actual sufferings by which the patient has been driven to seek aid. The more serious symptoms are often lightly touched upon, the more trivial exaggerated, and the whole jumbled together without logical sequence or the slightest attempt at orderly arrangement. This story, trying as it is to the physician, and all the more trying the more his own mind is duly trained, he ought to listen to; for this the patient expects, and perhaps has a right to expect. During the tedious narration it may give him patience to bear in mind two considerations: first, that from it he must obtain the right end of the clue which is to guide him in the difficult task of ascertaining the nature, extent, and seat of the disease; and second, that by this often most prolix narrative, taken along with his attitude, manner, and expression, the patient, absorbed in his own sufferings, is giving his physician, if he is careful and observant, the best opportunity of becoming acquainted with the *ego* with whom he has to deal.

The most critical examination of symptoms, the most careful inquiry into the state of internal organs, the most logical deductions from these as to the morbid changes from which they have originated, will often be erroneous unless the physician is also a student of human nature, and is able to arrive almost intuitively at some knowledge of the mental characteristics and peculiarities of his patient.

But sooner or later, and more often later than sooner,

the patient will have arrived at the end of his narration, and then the physician must unravel for himself this tangled web; and, taking the different threads, he must follow them up, and by means of close physical examination ascertain the condition of the various organs of the body—particularly those which the train of symptoms detailed indicate to be implicated in the morbid process. It is only by a methodical examination of the different systems of the body that a satisfactory view of the condition of the patient can be obtained, and the very foundation of rational treatment laid.

In the following pages an attempt will be made to explain the meaning and diagnostic significance of the chief symptoms and physical signs which are met with in disease. These group themselves naturally round the different physiological systems of the body—Alimentary, Absorbent and Hæmopoietic, Circulatory, Respiratory, Integumentary, Urinary, Reproductive, Nervous, Locomotory; and under these headings they will be considered.* This is not, of course, to be looked upon as a rigidly accurate division, but for practical purposes it suffices, and it has this great advantage—viz., that those who are habituated to follow such an arrangement in the examination of patients are less apt to neglect minute points which might otherwise escape the memory. Nor is it to be supposed that every patient requires to be subjected to so exhaustive a catechizing as this arrangement, if fully carried out, would necessitate. Many trivial complaints call for no such exercise of patience either on the part of the physician or on that of his patient, and in severe or urgent cases the first examination must necessarily be at best rapid and limited. Nor even where close inquiry is desirable is it necessary to follow accurately the sequence here given; and to some it may seem more suitable to clear up, first of all, the details regarding that system which seems most profoundly implicated, and only thereafter, and more cursorily, to examine into the condition of the others.

It must be carefully borne in mind that in examining a

* We follow in this respect the order of case-reporting which obtains in the wards under the care of the Professors of Clinical Medicine in the University of Edinburgh; and with this arrangement the author has long been familiar, as it closely corresponds to that which had been drawn up by Professor Grainger Stewart, and which was in use at the time the author was his Resident Physician in the Royal Infirmary.

patient we are dealing with a fellow-creature, and that all our inquiries and all our investigations must be conducted with the utmost courtesy, kindness, and patience.

In the following pages attention will first be directed to certain preliminary inquiries which should be made, and then to the various systems, in the order already mentioned.

CHAPTER I.

The General Aspect, Condition, and Circumstances of a Patient.

Preliminary Inquiries—Family History—Habits and General Surroundings at Home and at Work—Previous General Health—Origin and Course of Present Illness. Present Condition—Height and Weight—Development and Muscularity—Color of the Skin—Cutaneous Œdema and Emphysema—Perspiration—Expression of Face—Temperament—Attitude—Obvious Evidence of Previous Disease or Injury—Temperature.

Before entering upon the more minute examination of a patient, there are several more general and preliminary inquiries which should be made, and it is needless to say that the care and extent of the investigation required must depend on two factors: first, on its necessity, in view of the special disease present; and second, on the mental and bodily condition of the patient.

After noting the patient's name, age, occupation, residence, etc., it is well to record in as brief words as possible, and in his own language, his chief complaint. This is not to be in any sense a statement of diagnosis, but simply the patient's own impression concerning his case. Both in cases of phthisis and bronchitis, for example, we might be told that the patient sought advice on account of severe cough, and of this symptom we would make note as the most prominent in his own mind. We further ascertain, as closely as we can, the duration of the present illness, and record it briefly—so many days, months, etc., as the case may be.

Having thus formed in our minds a general idea, however ill-defined, of the case before us, we proceed to consider the

Family History.—Inquiry into the general health of the patient's family should be specially directed to ascertain whether any of his near relatives have suffered from those forms of disease which are usually supposed to be hereditary, such as consumption, scrofula, syphilis, rheumatism, gout, heart disease, and various nervous disorders. Such inquiry must not limit itself to the near relatives, father, mother, brothers, and sisters, but ought to extend to the aunts, uncles, and grandparents.

Habits and General Surroundings at Home and at Work.—Luxurious habits, "fast" living, and excesses of all kinds, are frequently the cause of disease, and any evidence of these must be sought for, and among them excessive alcoholic indulgence stands out prominently. To insufficient or unwholesome diet many ailments may be traced, as well as to long hours of work, and to the bad ventilation or defective drainage of the apartments used. It is also well known that certain occupations have a special tendency to produce disease, among which may be mentioned the pulmonary affections met with in miners, stone-masons, and cutlers, the anthrax which attacks wool-sorters, and the plumbism from which painters suffer.

We next proceed to inquire, with some minuteness, into the

Previous General Health.—Ascertaining the usual state of health, the date and nature of former ailments, liability to particular morbid conditions, present or previous residences, or other circumstances which may have influenced its production or development, exposure to contagion, etc.; and, if the patient be a female, it may be advisable to inquire into the condition of the reproductive functions.

Origin and Course of the Present Illness.—It is impossible here to do more than indicate certain general lines on which it is usual to proceed. Having already fixed the date of commencement of the illness, we would next endeavor to gain some accurate idea of the *manner* in which it commenced; with what symptoms; whether it came on suddenly or gradually; to what cause the patient traces his loss of health; and, if his statement does not appear probable to us, we must strive, by careful, guarded, and unobtrusive cross-examination, to satisfy ourselves on these points. Knowing the usual etiology of such a case as the

one we are studying, we possess a guide as to the direction in which our inquiries should be made. The sequence of symptoms may now be ascertained, the date of origin of each, and its severity; and, finally, we note to what medical treatment the patient has been subjected, and what was its result in his case.

PRESENT CONDITION.

Before proceeding to the examination of each system of the body; it is advisable first to note certain

General Facts.

1. **Height and Weight.**—In almost all diseases the weight becomes diminished, and in the course of treatment the patient should, when it is practicable, be weighed at regular intervals, when a very valuable indication of the progress of the malady will be in our hands. When, however, we have only the result of one weighing, it is of consequence to know what a man of a given height ought to weigh when in health. For this purpose, Mr. Hutchison has compiled a table (deduced from the examination of 3,000 persons), from which the following figures are taken:

A man of	4	ft.	6	in.	to	5	ft.	0	in.	ought to weigh about	92.26	lbs.
"	5	"	0	"		5	"	1	"	" "	115.52	"
"	5	"	2	"		5	"	3	"	" "	127.86	"
"	5	"	4	"		5	"	5	"	" "	139.17	"
"	5	"	6	"		5	"	7	"	" "	144.29	"
"	5	"	8	"		5	"	9	"	" "	157.76	"
"	5	"	10	"		5	"	11	"	" "	170.86	"
"	5	"	11	"		6	"	0	"	" "	177.25	"
"	6	"	0			.....				" "	218.66	"

2. **Development and Muscularity.**—To be typical of perfect health, the various parts of the body must be accurately proportioned one to another. A moderate amount of adiposity is quite consistent with health, provided that the muscular system is correspondingly developed. Generally, as age advances, the tendency to the deposit of fat increases, and this must be borne in mind. At the same time, its rapid accumulation, after fifty years of age, is not a symptom of health. Spare people are often the longest livers.

The presence of certain obvious morbid conditions may be noted at this stage.

3. **Changes in the Color of the Skin.**

(*a.*) *Pallor* is due to defective filling of the capillaries, to deficiency in the quantity of the blood, or of the hæmoglobin it contains. Pallor, consequently, may arise from any condition which prevents the proper assimilation of the food (dyspepsia, etc.); from any interference with the formation of the blood (chlorosis, anæmia, etc.); from any disease leading to loss of blood (hemorrhage), or its nutritive materials (Bright's disease), or, finally, from any affection of the vascular system interfering with the proper propulsion of the blood (mental emotions, fatty heart, mitral disease, etc.). Pallor occurs in connection with all grave organic diseases, such as cancer and tuberculosis. The paleness of the skin can best be appreciated, if it is slight, on the ears, cheeks, eyelids, or lips.

(*b.*) *Redness* of skin beyond the natural tint first, and principally, shows itself at those points which have just been mentioned in connection with pallor; but it must be borne in mind that those persons who are, by reason of their occupation exposed to heat, or to the weather, are usually ruddy in complexion. Apart, however, from these causes, redness occurs either as a result of increase of the amount of blood in the body, or of its hæmoglobin (as is seen in "full-blooded" plethoric persons), or is due to dilatation of the capillaries. The latter cause accounts for the blushing caused by mental emotion, as well as that following the inhalation of nitrite of amyl; and in a similar way may be explained the redness of the scalp and face in hemicrania, and the general redness of inflammation, and of fever.

(*c.*) *Cyanosis* (κύανος, blue), or blueness of the skin, varies much in degree. It is first noticeable on the lips, cheeks, conjunctivæ, ears, and point of the nose and fingers, but may become very general. It always arises from the presence of too much carbonic acid in the blood. This may be caused by interference either with the respiratory or with the vascular functions. Anything which prevents the free access of air to the blood circulating in the lungs will cause cyanosis, and hence this condition may arise in almost all laryngeal and pulmonary diseases. Cyanosis also attends diseases of the heart, whether congenital malformations or valvular affections; and may also be produced by spasm of the arterioles, as in chilling of the surface.

(*d.*) *Jaundice*, or yellow discolorations of the skin, results from the absorption of bile into the circulation. It shows

itself first under the conjunctivæ; but the observer must not mistake for jaundice the yellow color of small masses of fat which may be deposited there in advanced life. It is usual to divide jaundice into two varieties: (1.) *Hepatogenic*, or mechanical, which results from obstruction of the gall duct by catarrh, concretions, parasites, pressure of tumors, etc., from compression of the smaller branches of the duct in the liver substance, either from interference with the portal circulation, or in such diseases as cirrhotic, waxy, or fatty liver. (2.) *Hæmogenic*, arising from abnormal conditions of the blood itself. To this class belongs the jaundice which follows the inhalation of chloroform or ether, and possibly that met with in typhus, yellow fever, acute yellow atrophy of the liver, etc.

(*e.*) *Bronzing* occurs in Addison's disease (disease of the suprarenal capsules). Large tracts of the skin become bronzed, usually on face, neck, back of the hands, and specially where friction or pressure is most felt. The conjunctivæ are never discolored. Smaller and more sharply defined patches of pigment are sometimes seen in pregnancy, and in disease of the generative organs in the female. Local pigmentation is often the result of external irritation (blisters, etc.), and in *Pityriasis versicolor* the presence of the parasite *Microsporon furfur* gives rise to a brown discoloration.

(*f.*) *Gray* discoloration of the skin (argyria) occurs after the prolonged use of nitrate of silver.

(*Skin eruptions are noticed further on*).

Œdema arises from the collection of serous fluid under the skin. The swelling usually commences at the ankles —at first appearing only in the evenings, to disappear after the night's rest—it may increase to a very great extent, the limbs becoming much distended, pale, smooth, and glossy. The finger pressed upon the skin leaves a deep indentation when removed (pitting). In well-marked cases the external genitals become greatly swollen. Occasionally the œdema makes its first appearance in the lower eyelid, then usually arising from the acute inflammatory form of Bright's disease. Œdema may arise—

1. From obstruction to the return of venous blood to the heart. In this way it may occur in almost all diseases of the heart, and in some pulmonary affections, particularly emphysema. Pressure on the inferior vena cava in the

abdomen, from an enlarged liver or tumor of any kind, or on the veins of the leg may give rise to œdema; and local œdema is common round abscesses.

2. From alteration in the coats of the vessels arising from a blood supply defective in amount or in quality. Under this heading fall the so-called hydræmic dropsies seen in diseases of the kidneys, in anæmia, and in many wasting diseases.

Emphysema (Gr. ἐμφυσάω, to inflate) of the skin is produced by a collection of gaseous fluid in the subcutaneous cellular tissue. The skin pits very slightly on pressure, and there is a curious and unmistakable feeling of crackling under the finger. Apart from surgical causes, emphysema occurs as the result of loss of continuity in the air passages or at some portion of the alimentary tract. Ulceration or wound of the larynx or trachea may give rise to emphysema, but it more commonly results from rupture of one or more air vesicles in the lung. In the latter case the air is effused into the interlobular septa, and under the visceral pleura passes back to the root of the lung, thence to the mediastinal cellular tissue, and finally appears in the jugular fossa, and passes under the skin of neck and chest. Perforation of the œsophagus from whatever cause is liable to occasion subcutaneous emphysema, and ulceration of stomach or intestines may likewise allow of the escape of air under the skin if these structures have been previously glued to the abdominal walls by adhesions.

Perspiration.—Increased perspiration (hyperidrosis) may be general or local. In some nervous affections it has been seen to be limited to one lateral half of the body (hemidrosis). In very many diseases general hyperidrosis may be observed, and furnishes sometimes an indication of considerable importance. Such, for example, is the perspiration which occurs in almost all organic diseases in the stage of collapse, and that which follows the crisis of continued fevers (critical sweat). One of the stages of ague is that of profuse perspiration, and the night sweats of phthisis are among its most distressing symptoms. In acute rheumatism the skin is habitually moist, and usually whenever there occurs considerable dyspnœa, from whatever cause, it is accompanied by perspiration. It must not be forgotten that strong mental emotions (fear) may produce copious sweating.

Diminution of perspiration is seen locally in many chronic skin affections; and generally in almost all conditions of pyrexia, in diabetes, and sometimes in the cirrhotic form of Bright's disease.

Finally, the perspiration may be colored yellow from the presence of bile pigments in cases of jaundice, and in uræmia it may hold in solution large quantities of urea. In certain rare cases it has been observed to have a blue color (the cause of which is as yet doubtful), and still more rarely extravasation of blood on to the surface of the skin has been observed.

The Expression of the Face is often characteristic of the disease under which the patient is laboring. The pallor of anæmia and of carcinoma, the blueness of cyanosis, and the puffiness of renal disease have already been alluded to. In addition, we may mention the drawing up of the upper lip in acute peritonitis, the flush over the malar bones in cases of acute pneumonia and of hectic fever, the squint of hydrocephalus, and the prominence of the eyeball of Graves' disease. In the typhoid state the countenance is dull, motionless, and without expression, and the lips and teeth are covered with sordes; while in the stage of collapse of cholera, the face is sunken, wrinkled, the eyeballs retracted, and the skin livid. One more expression we may allude to—the *hippocratic*, that, namely, which immediately precedes death. The face is pale and leaden, the eyes sunken, the eyelids separated, the cornea less transparent than natural, the nose sharp and pinched, and the lower jaw falling.

Temperament, Constitution, or Diathesis.—Many persons show in their general appearance that they have a constitution which is liable to certain forms of disease. The recognition of such peculiarities of appearance may be of great importance in cases in which it is difficult or impossible to obtain satisfactory information regarding the family history. The more important of these varieties will here be sketched in outline.*

1. *The Sanguine Constitution.*—Body well developed, head

* For this outline the author is much indebted to the courtesy of Professor Grainger Stewart, from whose unpublished lectures it has been in part derived.

large, teeth massive and good, complexion ruddy, hair thick, digestion and nutrition good, pulse hard, blood pressure high. In later years the body becomes corpulent, and the signs of old age come on prematurely. Such persons are liable to arthritic affections of all kinds, and to diseases of the heart and blood-vessels (angina, fatty heart, aneurism, atheroma, apoplexy, etc.).

2. *The Nervous Constitution.*—Figure small and wiry, face mobile, features small and delicate, great activity of mind and body, dyspeptic, and with highly strung nervous system. Individuals of this temperament are specially liable to nervous diseases of all kinds.

3. *Strumous Constitution.*—The whole osseous system badly developed, joints enlarged, mucous membranes irritable, upper lip and alæ nasi thick, thorax contracted, skin pale and delicate, and hair thin. Such persons are liable to diseases of bones, lymphatic glands, and to tubercle in all its forms.

4. *Lymphatic Constitution.*—Body large and clumsy, muscles flaccid, face pale and expressionless, movements slow, and functions both of body and mind sluggish.

5. *Bilious Constitution.*—Face oval, long upper lip and chin, long nose, complexion dusky, digestion sluggish, liver in particular being inactive, nervous system not highly strung. In its further developments this constitution passes into melancholic, where the patient takes gloomy views of things in general. Such persons are liable to melancholic insanity.

6. *Gouty Constitution.*—Hair early gray, little tendency to baldness; nose short, rounded, and red; cheeks ruddy, eyes generally dark, teeth large and covered with thick enamel, digestion bad, suffers much from dyspepsia, heart tends to degenerative changes, arteries atheromatous, arcus senilis appears early and is well marked. Persons of this constitution are liable to all the forms of gout, to the cirrhotic form of Bright's disease, to neuralgia, and to apoplexy.

7. *Rheumatic Constitution* closely resembles the sanguine, as already described. There is, however, in it a greater tendency to fulness of body and less general vigor. The teeth are liable to early decay.

Of the other constitutions, such as the malarious, the hæmorrhagic, and the alcoholic, the limits of these pages do not allow allusion, nor need the numerous mixed forms be specially noticed.

Attitude.—The attitude of the patient is very frequently determined by the disease from which he suffers. For example, at the height of a severe fever, the patient may be seen lying on the back very flat, with the face turned upwards; and one of the first indications of improvement is his wish to be turned on to his side. The round-shouldered appearance of the asthmatic and emphysematous is characteristic; and in bed those who suffer from dyspnœa are usually obliged to be propped up. Again, a patient suffering from acute peritonitis lies with his knees drawn up; and so with all diseased conditions, almost every one of which compels the assumption of some more or less characteristic attitude.

Evidence of Previous Injury or Disease.—Apart from the obvious traces of such surgical affections as fractures, etc., it is always of importance to note any indications of previous disease which may meet us in the course of our examination. The pitting of smallpox, the cicatrices over scrofulous glands, the large joints of those who, in their childhood, have been subject to rickets, old-standing paralyses, are among these indications. Perhaps the most important are the signs of former syphilis, such as fissures at the angles of the lips, cicatrices on the palate, adhesions of the iris, traces of skin eruptions, nodes on the skull or tibiæ, etc. Congenital syphilis may be recognized late in life by various indications, among which are pallor, depression of the bridge of the nose, pegged teeth, prominent forehead, deafness, nodes on bones, keratitis, cicatrices of old ulcers, falling out of hair on head and eyebrows, etc.

Temperature.—The thermometer used for estimating the temperature of the body, ought to be of such a size, and so divided that tenths of a degree can easily be read off; it ought to have an arrangement for maximum registration; the purchaser of a clinical thermometer should see that the accuracy of the instrument in question has been tested by comparison with a standard thermometer.

It is most usual to take the temperature by placing the instrument (the index having been first jerked down below the normal point) in the axilla, the skin of which region must be carefully dried, should there have been any perspiration. To give reliable readings, the thermometer

ought to remain in position for fifteen to twenty minutes, and during the last five minutes its height should be several times examined, in order to see if it remains stationary, or, not having reached its highest point, is still rising. In the latter case it ought to be allowed to remain longer, until no farther elevation takes place. Still more accurate results are to be obtained, and in shorter time, when the instrument is passed into the rectum. In children the temperature is more readily taken in the mouth, the bulb of the instrument lying under the edge of the tongue.

In all cases of importance the temperature ought to be taken twice a day, morning (9 to 11 A.M.) and evening (5 to 7 P.M.), and in serious cases every two hours. From the data so obtained charts should be constructed in the usual manner.

While the *mean normal temperature* may be taken to be 98.6° of Fahrenheit's scale, yet variations from 97.5° to 99.5° are met with in healthy persons. When the mercury stands below the former point, however, it usually indicates a state of collapse. Above 99.5° the thermometer indicates a condition of fever, which, when it surpasses 105°, is spoken of as hyperpyrexia. More important for diagnosis than the actual reading is the manner in which the temperature rises. Four types of temperature curves are to be distinguished.

1. *Continued.*—The temperature rises rapidly and almost continuously to a given height, at which it remains during the time the fever lasts, subject only to such slight differences between morning and evening temperature as may be observed in health, and then it falls to, or even below, the normal point, as suddenly as it rose. This rapid fall, which is coincident with the commencement of convalescence, is denominated *crisis*. This type is met with in typhus, smallpox, measles, etc., but is perhaps best marked in acute lobar pneumonia.

2. *Remittent.*—In this variety the difference between the morning and evening temperatures is considerable (say 2° F. or more). Hectic fever in all its forms is perhaps the the best example, and it is to be noted that occasionally (especially in phthisis) the ordinary state of matters is reversed, the morning temperature being high, and the evening low. During the first few days of typhoid fever the temperature rises in a characteristic manner, each morning remission being considerably under the temperature of the

preceding night, but above that of the preceding morning (In typhus fever, as a rule, the rise is more sudden.) In well-marked examples of this type of fever temperature, when the disease takes a favorable turn the re-establishment of the normal temperature takes place very gradually, and the slow fall of temperature is called *lysis*, to distinguish it from the more rapid crisis of which we have already spoken.

3. *Intermittent.*—This type is seen in the different forms of ague. The temperature rises with great rapidity to a very high point, and falls again in a few hours to normal. There then follows a period of intermission, when the condition of the patient is normal, and then again, the temperature rises as before, and so on. The intervals vary in duration according to the type of ague present, lasting twenty-four hours in quotidian, forty-eight in tertian, and seventy-two in quartan ague.

4. *Relapsing or Recurrent Type.*—After a rigor the temperature rises to a high point, near which it remains for some days, and then falls to normal (crisis). An interval of immunity from fever of varied duration (five to eight days or longer) succeeds this attack, and then follows a relapse which corresponds very closely to the original attack. These relapses may occur several times before the disease ceases.

CHAPTER II.

Alimentary System.

Section I.—Objective Phenomena:—Lips—Teeth—Gums—Tongue—Secretions of Mouth—Fauces—Mastication—Deglutition—Examination of Œsophagus. Section II.—Subjective Phenomena:—Appetite—Thirst—Sensations during Fasting—Sensations after Eating—Pain—Acidity — Heartburn — Water-Brash — Flatulence — Eructation. Section III.—Excretory Phenomena:—Nausea and Vomiting—Examination of the Vomited Matter—Defæcation—Examination of the Fæces.

The signs and symptoms in connection with the alimentary system may be conveniently considered under two sections. The first of these contains the various objective

phenomena which present themselves during the examination of organs of mastication and deglutition, the subjective sensations of digestion, and the phenomena connected with the expulsion of the food from the body, either by vomiting or by defæcation.

The second group includes all these physical signs which the examination of the abdomen affords.

The first of these sub-divisions will be treated of in the present chapter.

Section I.

Lips.—In examining the lips we have to note—

(*a.*) *Color.*—Owing to the great transparency of the labial epithelium, any change in the color of the blood circulating in the minute vessels underlying it can be readily distinguished. When the lips assume a dusky-blue cyanotic* color, we know that the circulation is being carried on imperfectly, which may be due to defective circulation through the lungs, or to interference with the entrance or exit of air to or from the pulmonary alveoli, or to a combination of these two causes. The lips may have a pale waxy color, indicating that the amount of hæmoglobin is either absolutely less than normal, as in cases of loss of blood, or is both absolutely and relatively (in relation to the aqueous constituents of the blood) below the normal standard, as, for example, in chlorosis and other forms of anæmia, as well as in many cases of Bright's disease.

(*b.*) *Form.*—Abnormal thickness or thinness of the lips gives an indication of the amount of the serum contained in the interstices of the tissues. In cases where this is abnormally great, without, however, any appreciable œdema (such as is met with in the early stages of Bright's disease), the lips are thick and pallid. The thin pinched lips which are seen, for example, in the second stage of cholera, indicate an abnormal diminution of interstitial lymph. Herpetic eruptions upon the lips (herpes labialis) occur in feverish conditions, particularly in pneumonia; and in syphilis deep and painful fissures are often met with. When we find the lips dry, cracked, and coated with sordes, as in most febrile conditions, we know that the patient has been breathing through the mouth, this causing (in combination

---

* From the Greek κυανος, blue.

with the raised temperature) an abnormally rapid evaporation of the saliva which in health keeps the lips moist.* The result of this rapid evaporation is that the solid constituents of the saliva are deposited on the lips in the form of sordes, whilst the cracking is due to the unequal contraction of the epithelial layer in the act of drying. The fact of the mouth being kept constantly open for the purpose of respiration, although frequently due to more or less complete obstruction of the nasal passages, may also result from any cause which interferes with the proper oxygenation of the blood; the natural tendency in such cases being to attempt to increase the quantity of air respired. All dyspnœic patients, therefore, tend to breathe through the mouth, as offering a freer passage for the ingress of air than the nostrils. The fur on the lips occurs under similar conditions as does that upon the tongue, which will be presently described in detail.

(*c.*) *Movements.*—The lips being among the principal organs of expression of the emotions and the will, many affections of the central nervous system lead to trembling of the lips (as for example, delirium tremens). Abnormal contraction and relaxation of the labial muscles usually mimics and exaggerates some normal expression. The *risus sardonicus* of tetanus and of strychnine poisoning may be taken as examples of this. The opposite condition is seen in the expressionless appearance which occurs in double facial paralysis.

Teeth.—The two sets of teeth usually appear in the following order:

MILK TEETH.		PERMANENT TEETH.	
Central incisors,	7th month.	Anterior molars,	7th year.
Lateral incisors,	9th "	Central incisors,	8th "
First molars,	15th "	Lateral incisors,	9th "
Canines,	18th "	Anterior bicuspids,	10th "
Second molars,	24th "	Posterior bicuspids,	11th "
Usually the lower central incisors appear first, then the upper central incisors, and after them the lower lateral incisors.		Canines	12th "
		Second molars,	12th to 14th "
		Third molars,	18th to 25th "

It is important to observe the *shape* of the teeth. In congenital syphilis the central incisors, usually of the upper

* The amount of saliva secreted is also diminished in fever.

jaw (permanent teeth), are very often considerably altered, as was originally pointed out by Hutchinson. They are shorter and narrower than natural, notched at the lower edge, and sometimes grooved down the centre, and set at greater intervals in the gums than normal.

Caries of the teeth must be noted as bearing upon neuralgia, dyspepsia, etc. In young and otherwise healthy subjects the usual cases of extensive caries of the teeth is acidity of the buccal saliva, arising from a slight catarrhal condition of the mucous membrane of the mouth. The continued and excessive use of mercury gives rise to a looseness of the teeth.

Gums and Mucous Membrane of the Cheeks.—Like the lips, the gums show by an anæmic, bloodless appearance the presence of defective circulation, or diminution of hæmoglobin in the blood. In chronic lead poisoning a *blue line* forms on the gums close to the dental margin, caused, in all probability by deposit of sulphide of lead, precipitated there by the sulphuretted hydrogen generated by the decomposition of particles of food remaining about the teeth. Swelling and tenderness of the gums, along with the looseness of the teeth already noted, are among the earliest signs of the action of mercury. Spongy gums which readily bleed are met with in cases of scurvy. Inflammation of the gums is either general (stomatitis), which may be of parasitic origin (muquet or thrush), or local, constituting gum-boil. Bleeding may also result from the sharp angles of carious teeth, or from the coming away of tartar from the teeth, which lacerates neighboring soft parts. In paralysis of the buccinator muscle the cheek hangs loose, and the food collects between it and the teeth. This condition is seen in facial and bulbar paralysis.

Tongue.—In examining the tongue three distinct points have to be considered—(1) its form, (2) its movements, (3) the condition of its surface.

(1.) *Form.*—In health the tongue varies much in shape, and this without any particular significance. It becomes swollen from various causes, particularly inflammation, the result of smallpox or scarlatina, or the abuse of mercury or other drugs, and from the presence of cancerous or syphilitic new formations. In dyspepsia the tongue has frequently a swollen, sodden character, marked at the edges by the

teeth against which it has been pressed; and even without these causes, where there is great mental hebetude along with defective movement of the organ in the mouth, such œdematous swelling takes place.

(2.) *Movements.*—The tongue receives its motor innervation through the hypoglossal nerve. Spasm of the lingual muscles is a common symptom in chorea, hysteria, eclampsia, epilepsy, etc. In chorea the tongue is protruded rapidly, and as rapidly withdrawn. Tremor of the tongue is a symptom of progressive muscular atrophy, generally paralysis, and bulbar paralysis, and is seen in delirium tremens.

Paralysis of the tongue is frequent in cerebral affections (hemiplegia from hemorrhage, embolism, etc.; bulbar paralysis, general paralysis, the advanced stages of locomotor ataxia, etc.). In unilateral paralysis the tongue when protruded is inclined towards the diseased side. When the paralysis is bilateral the tongue is relaxed, wrinkled, fibrillary contractions may be seen on its surface, and the speech becomes inarticulate and unintelligible.

(3.) *Surface of the Tongue.*—Note first its condition as to moisture. Whenever a patient breathes habitually through the mouth * there is a tendency to dryness of the tongue, as well as of the lips (as has been already stated), because of the more rapid evaporation of the saliva and buccal mucus, which usually keep these parts moist. In fever this is still more marked, partly because there is then diminished secretion of these fluids, and partly because the evaporation goes on more quickly on account of the elevation of the temperature. In order to prevent this dryness the patient moistens his lips with water at very short intervals, and hence the dry tongue is not always present even in fever. When it is present in such patients it shows that thirst is not being felt, and is an indication that the senses are becoming blunted and consciousness is being lost. When the tongue becomes very dry, cracks appear on its surface, due to the unequal contraction of the epithelium in the act of drying. A degree of dryness of the tongue is likewise met with in diabetes, and after the administration of certain drugs—atropine, for example.

---

* Either on account of obstruction of the nasal passages or from anything which interferes with the proper oxygenation of the blood (dyspnœa in all its forms).

Fur on the Tongue.—In fever when, as has just been said, the saliva evaporates quickly, it deposits its solid constituents upon the lips and tongue. When, however, no abnormal evaporation is taking place, the fur which is found upon the tongue is not to any appreciable extent so formed. It then consists of *débris* of food, of cast-off epithelial cells, and of masses of micro-organisms.* These organisms enter the mouth in the air or in the food, are caught on the filiform papillæ, to which they firmly adhere, and there multiply with great rapidity. The fungiform papillæ are too smooth to afford points of attachment, and hence they are usually free from fur, and stand out red and prominent. In scarlet fever these papillæ are more than usually conspicuous, on account of the congestion of the mucous membrane, and they stand out distinctly through the creamy fur which covers the rest of the organ, and hence the tongue assumes a "strawberry" appearance. The fur which collects on the lingual surface is being constantly detached by the rubbing of the tongue against the roof of the mouth, gums, and teeth; and as during sleep there is very little movement, the coating is always thickest in the morning. It is also to be remarked that the fur collects most where the tongue is roughest, and where the movement is least in amount, *i.e.*, the centre and back part. Increase of fur may result from any condition which diminishes the movements of the tongue on the palate and gums, such as dryness of the mouth, swelling of the tongue, defect of the palate, or disease of the central nervous system (hemiplegia, bulbar paralysis, etc.) paralyzing the lingual muscles.

But in addition to the fur which adheres to the tongue, the cavity of the mouth often affords a nidus for other forms of vegetable organisms, such as those which give rise to lactic and butyric fermentation of sugar, and acetous of wine. By far the most important of these is the parasite which occasions thrush, the so-called *Oidium albicans*, which Grawitz has lately shown † to be identical with the *Microderma vini*. Thrush is usually met within delicate children, and affects the inner surface of the lips, and the

---

* According to Butlin's cultivation experiments (St. Bartholomew's Hosp. Rep. xv.) these include: *Micrococcus*, *Bacillus subtilis*, *Sarcina ventriculi*, *Spirochæte Obermeyerii*, *Bacterium termo*, and a form of *Vibrio*.

† Virch. *Arch.*, vols. lxx. and lxxiii.

mucous membrane of the mouth generally. Small white points first form, which rapidly increase in number, and extend in area, the patches appearing like curdled milk. The reaction of the saliva becomes strongly acid, and thus causes intense irritation of the mucous membrane of the mouth, which becomes red, swollen, and painful. In adults the appearance of thrush is usually of ominous import.

The color of the tongue varies considerably under different conditions. In fever it is generally more or less reddened, and it may become blue in cyanosis, or pale where anæmia exists.

Finally, the tongue is liable to be the seat of new formations—syphilitic fissures, ulcers, and deposits are frequently met with, and cancerous disease of that organ is not uncommon.

The sense of taste will be considered under the nervous system.

Saliva.—The *reaction* of the mixed saliva found in the mouth is normally alkaline, but it becomes acid when retained for long in the buccal cavity, in dyspepsia, and in diabetes. It is mixed with epithelium and leucocytes, and often contains certain vegetable organisms (*Oidium*). A small quantity of albumen is present in health, and may be much increased in disease.* Certain medicines when administered internally appear in the saliva. In particular, the salts of iodine and bromine; urea is also sometimes present in cases of uræmia, but bile and grape sugar are never excreted in the saliva. In disease the saliva may be increased or diminished.

Increase of saliva (salivation or ptyalism) arises—

1. From irritation in mouth and throat; as in stomatitis (mercurial or simple), gum-boil, ulcers, dentition, sore throat, etc.

2. From irritation in stomach, pancreas, intestines, uterus; as in cases of dyspepsia, worms, and in pregnancy.

3. From neuralgia, especially of the face.

4. From certain diseases of the brain, medulla, and spinal cord. In insanity, hydrophobia, hysteria, and particularly in bulbar paralysis.

---

* In catarrh of the mucous membrane of the mouth, the buccal mucus contains large numbers of epithelial cells and leucocytes and a great quantity of dissolved albumen.

5. From the action of certain drugs, in particular, mercury and jaborandi.

Diminution of saliva is chiefly met with in fevers and in diabetes. It occasionally results from blocking of the salivary duct with a calculus. It may also be diminished by mental emotions, and by the administration of certain drugs.

Fauces.—To examine the fauces the patient must be made to open his mouth in such a position that strong light falls on the back of the throat. In most cases it is necessary to depress the tongue by means of a spatula or large spoon. Having obtained in this manner a satisfactory view, we must satisfy ourselves of the condition of the pillars of the fauces, with the tonsils lying between them (in health barely visible); of the soft palate arching upon either side, with the uvula depending from the centre; and of the posterior wall of the pharynx behind. We must look for enlargement, inflammation, or ulceration in all these parts, and for changes in the mucous membrane covering them. Acute catarrh causes redness and swelling of the fauces; and to a still more marked extent does the inflammation which accompanies scarlet fever. More chronic inflammatory conditions give rise to a dark-red appearance of the mucous membrane, in which the follicles may be more conspicuously affected; and care must be taken not to mistake the distended mouths of follicles for ulcers. In thrush (muquet) small white patches like curdled milk are scattered over the fauces, as well as over the mouth generally; and in these, when microscopically examined, filaments and spores of *Oidium albicans* can be seen. In acute tonsillitis the tonsils are much swollen, and of a dark-red color. Their surface is nodulated, and covered with mucus. When the disease becomes chronic, the tonsils are frequently marked with small white points of sebaceous matter projecting from the follicles.

Ulceration of the throat may be (1) catarrhal; (2) follicular (corresponding to the follicles); (3) scarlatinal; (4) syphilitic; (5) diphtheritic; (6) herpetic; (7) cancerous; (8) result of the action of hot water, of acids, alkalies, or other chemical substances. In diphtheria the mucous membrane is swollen, reddened, and covered with patches of a grayish exudation, which become matted together so as to form a false membrane. These patches are most frequently met

with on the soft palate and uvula, but often extend to the tonsils, pharynx, etc. The mucous membrane, in which this exudation is embedded, sloughs and separates, leaving open ulcers. Abscesses may form in the tonsils, or behind the posterior wall of the pharynx. In the latter case, the pharyngeal wall bulges forward, interfering alike with respiration and with deglutition. Such retropharyngeal abscesses usually arise from disease of the cervical vertebræ, the condition of which must be carefully examined. Very rarely carotid aneurisms may bulge in this direction.

The form of the soft palate is of considerable importance in diagnosis. Paralysis may be partial or total. In the former case the velum droops and does not arch when the uvula is tickled, and the uvula is usually, though not always, bent to the sound side.* This condition is caused by paralysis of the levator palati and the azygos uvulæ, both of which muscles receive their nervous supply from the facial nerve through the large superficial petrosal branch and Meckel's ganglion; and as this nerve leaves the facial at the geniculate ganglion, paralysis of one side of the palate shows that the disease of the seventh nerve is above that point.

When all the muscles on both sides are paralyzed, the soft palate becomes loose and flapping, the speech assumes a peculiar nasal tone, and fluids regurgitate through the nose when the action of swallowing is attempted. This total paralysis of the palate occurs in bulbar paralysis, and in various affections of the brain and spinal cord. Paresis of the muscles of the palate frequently arises after diphtheria.

Mastication may be rendered difficult or painful by the presence of inflammatory affections of the lips, gums, cheeks, or tongue, by defective teeth, by cancerous or other ulceration of those parts, or by paralysis or spasm of the muscles employed in the act. The buccinator and the orbicularis oris receive their motor supply from the seventh nerve; and when that nerve is paralyzed the food accumulates between the cheek and the teeth. The motions of the tongue are affected in paralysis of the hypoglossal nerve. The muscles of mastication (masseters, pterygoids) are innervated from the motor branch of the fifth nerve, and

* Sanders, *Edinburgh Medical Journal*, 1866.

when they are paralyzed the lower jaw hangs down, and proper mastication is impossible.

Deglutition may be rendered difficult and painful by various affections of the mouth and tongue, such as swelling, ulceration, inflammation, etc., but apart from such temporary causes it may arise from paralysis of various groups of muscles. For purposes of description the act of deglutition may be divided into three stages: (1) The gathering up of the food into a bolus and thrusting it through the anterior pillars of the fauces. (2) Its passage through the upper part of the pharynx until it has passed the orifice of the larynx. (3) Its descent through the lower part of the pharynx and the œsophagus. The first stage of deglutition is interfered with in paralysis of the hypoglossal nerve, for the tongue cannot then form and force back the bolus into the fauces. Such paralysis is almost invariably of central origin. In the second stage of deglutition, disease of the facial nerve above the geniculate ganglion, by causing paralysis of the soft palate, allows food to pass into the posterior nares, and the same symptom is present in post diptheritic paralysis, in syphilitic ulceration of the palate and epiglottis, and in cleft palate. This second stage of swallowing may also be interfered with by paralysis of the muscles of the pharynx. The food sticks at the root of the tongue, and occasions such dyspnœa as to require its removal by means of the finger, and fluids pass readily into the larynx. Thus paralysis, rare as a peripheral disease, is common as a result of affections of the pons and medulla, and of diseases of the base of the brain, compressing the cranial nerves. The third stage of deglutition may be interfered with by mechanical obstruction of the œsophagus (impacted foreign bodies, pressure of aneurism or other tumor, simple or cancerous stricture, etc.), or by paralysis of the muscles of the œsophagus, which is very rare as an isolated affection. In the latter case solids may manage to make their way down the tube by their own weight. Spasm of the œsophageal muscles sometimes prevents deglutition in cases of hysteria.

Examination of the œsophagus is practically limited to mediate palpation by means of the œsophageal bougie. It is most convenient for the purposes of diagnosis to use the elastic tube of a stomach pump. The tube must be softened in warm water, lubricated (preferably with glycer-

ine or white of egg), and passed gently over the back of the patient's tongue, being guided by the left forefinger of the operator over the epiglottis, and then gently and steadily pushed into the stomach. Of the difficulties and dangers which the operator may meet in the course of this exploration, the following are the most important: The œsophagus must never be sounded until there is certainty that no aneurismal tumor is pressing upon it, into which the point of the tube might be forced. In ordinary circumstances there is but little danger of the instrument passing through the glottis into the trachea, but whenever paralysis and anæsthesia of the laryngeal structures exists very great care must be taken. It is hardly necessary to warn the student of the danger of forcing the sound through the œsophageal structures. Such an accident has happened not infrequently, and it is of course more ready to occur when the walls are softened by cancerous deposit.

In sounding the œsophagus we meet with—

1. *Pain.*—If the pain be felt again and again at the same spot it indicates a local process, probably of an inflammatory nature. The presence of ulceration may be conjectured if the sound, however carefully it has been introduced, always comes away smeared with blood.

2. *Obstruction.*—This may be caused by the point of the sound passing into a diverticulum, and it is characteristic of this condition that the instrument sometimes passes with great ease, and sometimes is absolutely arrested. Strictures of various kinds also prevent the passage of the sound. The purely spasmodic strictures met with in hysteria may be distinguished from those due to organic disease by placing the hysterical patient under the influence of chloroform, when an instrument which before was obstinately resisted now passes freely into the stomach.

Auscultation of the œsophagus is occasionally practised. The stethoscope is to be applied a little to the left of the spinal column in the cervical or dorsal region, and the sound listened to which arises in the œsophagus when the patient swallows water. In health the act of deglutition is accompanied with a short, clear gurgling sound. When, however, œsophageal obstruction exists, this sound is prolonged, and altered in character below the seat of the stricture.

Appetite.—Derangements of the appetite may occur both

in general diseases and in local affections of the stomach and intestines.

Anorexia, or loss of appetite, is present during all acute febrile diseases, and may also result from excessive fatigue of mind or body, or from depressing emotions, such as pain or grief, as well as from the use of narcotics or alcohol. It is also caused by inflammatory affections of the stomach, cancer of the gastric walls, constipation, and other abnormal conditions of the intestines.

Boulimia, or excessive appetite, may result simply from the habit of over-eating, or may be due to the presence of worms in the stomach or intestines. It is also present in certain chronic imflammatory conditions of the gastric mucous membrane, and is a prominent symptom in the course of diabetes, and in various nervous disorders (insanity, hydrocephalus, epilepsy, hysteria, and hypochondriasis).

Pica, or depraved appetite, in which the patient craves for various abnormal and even disgusting articles, is sometimes met with during pregnancy, and in patients suffering from chlorosis and mania, and in idiots.

Thirst almost invariably attends all feverish states of the system. It is a marked symptom in diabetes; and in irritative conditions of the stomach thirst commonly appears some hours after eating.

Sensations during Fasting.—In the atonic form of chronic dyspepsia there is frequently before meals a feeling of sinking in the epigastrium, along with faintness. Pain, when the stomach is empty, may sometimes be present, due to cancer or ulcer, although in these diseases the greatest pain is usually after food has entered the stomach. Over-secretion of gastric juice may give rise to pain when the stomach is empty, which is then usually relieved by eating.

Sensations after Eating.—Painful sensations after food is swallowed, which are referred to the gastric region, vary from the slightest feeling of discomfort or oppression up to the most severe agony. The pain may be due to—

1. *The Presence in the Stomach of Irritating Substances.*—This irritation may be mechanical, when indigestible food has been swallowed, or chemical, from the presence of corrosive poisons. In its slighter forms such irritation gives rise merely to a feeling of weight, discomfort, or distention; in its graver varieties the pain is very great.

2. *Organic Diseases of the Gastric Wall.*—Of these the most important are cancer and gastric ulcer. In both of these affections, especially in the latter, pain becomes very severe almost immediately after food has been swallowed, and continues for a considerable time, though, as a rule, the patient is free from pain when the stomach is empty. In gastric ulcer the pain is sometimes sharply localized, and is increased by external pressure. Extensive organic disease may, however, in rare cases, run its course without pain.

3. *Abnormalities of Secretion.*—Acidity, when due to over-secretion of the acid element of the gastric juice, or to fermentation of the partially digested contents of the stomach, may give rise to pain. In the latter case the evolution of gas, by causing distension of the walls of the stomach, or spasmodic contractions due to the irritation of the gastric mucous membrane, may be the direct factor in producing the painful sensations. The name *heartburn* has been given to a peculiar pain of a burning character, due to abnormal acidity of the contents of the stomach. It is referred to the epigastrium, and spreads upwards to the pharynx.

4. *Nervous Causes.*—Pain in the stomach is occasionally of a purely neuralgic character, occurring most usually in chlorosis, hysteria, hypochondriasis, and perhaps also in gout. In such cases the digestion may be perfect. The pain is frequently relieved by firm pressure, resembling in this respect the pain of flatulence.

Pain in the abdominal muscles, and in the transverse colon, must not be mistaken for gastric pain.

Acidity.—Acidity manifesting itself by pains (heartburn in particular), eructation of gas, and sometimes by nausea and vomiting, may arise from various causes.

1. *Over-Secretion.*—Some observers are of opinion that in certain nervous conditions a secretion of gastric juice greater than normal is produced, as in "nervous dyspepia," * or as a result of irritation of the gastric mucous membrane from cancerous growths or ulcer; and that in this way acidity may develop itself.

2. *From Fermentation.*—In the normal condition no fermentation can take place in the contents of the stomach, on account of the strongly acid reaction which they possess;

* It appears probable that in these cases there is a certain amount of gastric congestion and subacute catarrh.

but whenever this acidity is sufficiently diminished fermentation may set in, and acetic, butyric, and lactic acids are then formed, along with hydrogen and carbonic acid gas. This condition of the gastric contents favorable to fermentation may arise from a deficiency of the gastric juice in quantity or quality, or from the introduction of large quantities of fermentable food. To aid in distinguishing the acidity from over-secretion from that caused by fermentation, it may be borne in mind that the former occurs in an empty stomach or immediately after food, whereas the latter does not attain its height until some hours after a meal, and does not occasion so much pain. The fluid (water-brash) regurgitating may be bland, neural, and tasteless.

Flatulence.—The collection of gas in the stomach and bowels may result either from the swallowing of air or from fermentative changes. It usually arises as a result of perversion of the digestive process in some of its various actions. When it reaches a considerable degree it is called tympanites or meteorism, and its seat, whether in the stomach or the bowel, may be determined by percussion, as will be afterwards explained. Flatulence is frequently accompanied by colic, and the gas is expelled either through the rectum or mouth. Gaseous eructations have been analyzed at various times, and have been found to contain carbonic acid gas, hydrogen, marsh gas, olefiant gas, oxygen, and nitrogen.*

Nausea and Vomiting.—The expulsion of the contents of the stomach in vomiting is caused by the forcible contraction of the abdominal muscles, diaphragm, etc., the gastric muscular fibre itself remaining inactive. It is a reflex act, having its centre in the spinal cord. This reflex centre corresponds apparently to a considerable portion of the cord, and includes part of the respiratory centre. Vomiting is produced in various ways: (1) By any disease of the brain or spinal cord which involves this reflex centre; (2) By excessive irritation of the respiratory centre, in violent coughing, etc.; (3) By the presence in the blood of any substances which occasion irritation of the centre or of the terminal twigs of the vagus, such as effete products in

* *Vide* analysis by Ewald and Rupstein, *Arch. f. Anat. u. Physiologie*, 1874.

uræmia, emetics, etc.; (4) By irritation of the vagus or its branches. This is by far the most frequent cause of vomiting. It is thus that vomiting is to be explained in cases of uterine disease or pregnancy, kidney disease, the passage of gall-stones, tickling of the soft palate, and, above all, in affections of the stomach itself. Chemical or mechanical irritation of the gastric mucous membrane gives rise to vomiting. It may also be produced by fermentative changes in the contents of the stomach, by ulceration, or chronic catarrh of the gastric mucous membrane. There is no disease of the stomach in which vomiting may not be present. Gastric vomiting is usually preceded by nausea and pain, and bears some relation to the food swallowed; whereas when this symptom arises from irritation of other kinds these are frequently absent. The diagnostic significance of vomiting can, however, be best appreciated after the examination of the

Vomited Matter.—This consists of the contents of the stomach and sometimes of the duodenum. The fluids and solids vomited are more or less acted upon by the gastric juice, and are usually strongly acid. Of abnormal substances present the most important is *blood*, which may be almost pure, but is generally more or less acted upon by the gastric juice, and thereby coagulated and blackened so as to resemble the grounds of coffee. Hæmatemesis (the vomiting of blood) is most frequently produced by gastric ulcer; but it also occurs in blood diseases (yellow fever), in congestion of the veins of the stomach (cirrhosis of the liver, pressure on inferior vena cava), and sometimes vicariously, when the menstrual flow is arrested, and finally, it may result from wounds of the stomach, or from the bursting of an aneurism into the stomach or œsophagus. Bile is frequently found in the vomited matter, rarely pus, and still more rarely fæcal matter. The latter usually points to intestinal obstruction, although, according to Bamberger, it may occur independently of such condition when the bowel is paralyzed as a consequence of peritonitis and typhoid fever. *Microscopic examination* of the vomited matter shows the various substances derived from the food swallowed, sometimes *Sarcinæ ventriculi* and intestinal parasitic worms (*Ascaris lumbricoides*, etc.) The *sarcinæ* are small cube-shaped cells united in groups of four. They are met with in nearly all cases in which food remains too

long in the stomach and there ferments, particularly in dilated stomach. Other micro-organisms may also be found, but are of less importance, so far as our present knowledge goes.

Defæcation.—While normally defæcation occurs once in the twenty-four hours, it is not uncommon to find persons who have two motions in that period, or whose bowels act only once in two days, without the bounds of health being overstepped. In infants the bowels move frequently—four or five times a day. The following points have to be inquired into:

1. *The Frequency of the Motions*, and the period at which the bowels act relatively to eating, drinking, exercise, etc.

2. *The Character of the Act of Defæcation.*—Faintness or sickness may precede the act, which may itself be painful and straining, and may be followed by a sensation of the rectum not having been emptied of its contents (tenesmus). The actual condition of the anus and rectum must be determined, and the presence of piles, fissure, prolapse, ulceration, etc., looked for.

The two conditions of constipation and of diarrhœa demand brief notice.

Constipation may result from—

1. Mechanical obstruction; and this may be caused in various ways, such as from accumulations of various kinds in the bowel, by cicatricial or cancerous stricture, by external compression of the intestines, by strangulation or intussusception, and by spasm or paralysis.

2. Defective peristaltic action.

3. Deficiency of the secretions.

These two last causes may arise from too frequent use of purgatives, from neglect of the regular performance of the act of defæcation, from the abuse of opium, from sedentary and from enervating habits, as well as from derangement of stomach and liver, and from many other causes too numerous to mention.

Diarrhœa.—Among the causes of this condition are—

1. Irritation of the bowels by improper or badly-digested food, by purgatives, etc., or by retained hardened fæces, or the presence in the bowels of noxious materials eliminated from the body, in various diseases, as uræmia, gout, and fevers.

2. Diseases of the bowels, congestion, inflammation, etc.

Character of the Fæces.*

1. *Macroscopic Characters.*

(*a.*) *Color.*—The normal color of the fæces may be altered by reason of the food eaten, and still more markedly by medicines. Iron and bismuth when taken internally blacken the motions, while the administration of preparations of iodine causes a blue, calomel a green, and logwood a red-brown color to appear in the fæces. If all these causes of altered color be excluded, then the alteration is dependent upon bile or upon blood. The presence of bile gives rise to a yellow or to a green tint, and in its absence the fæces assume a gray or chalky appearance. Blood in the stools may possess its natural appearance, in which case it has probably come from low down in the intestinal tract, and is in consequence not intimately mixed into the substance of the fæces, but merely lies on the surface. If, however, the bleeding point lies higher up, then the blood becomes acted upon by the digestive fluids, and assumes a dark-brown or black appearance in the stools (melæna), with the substance of which it is intimately mixed.

(*b.*) *Reaction.*—According to the exhaustive investigations of Nothnagel, the reaction of the fæces is variable, but is usually alkaline, almost always so in typhoid fever. In the acute catarrhal enteritis of children, the stools are usually acid.

(*c.*) *Form and Consistence.*—The fæces become fluid when the intestinal peristaltic action is accelerated, and on the contrary in constipation they may assume a very considerable hardness. The presence of a polypus in the rectum may impress a longitudinal groove upon the fæcal masses; and when there is narrowing of the bowel, particularly of the rectum, the fæces are usually thin, long, and narrow.

(*d.*) *The Odor* of the fæces has not been shown to be of diagnostic significance.

(*e.*) *Abnormal Substances visible to the naked eye.*—Either on account of their own indigestibility, or from interference with digestion, various articles of food may appear in the fæces. Of importance is the detection of gall stones, and when their presence is suspected, the fæcal matters should be carefully washed on a fine sieve. Shreds of mucous membrane, polypi, and other tumors, are occasionally met with, and more frequently still the various parasites that

* See Appendix A.

inhabit the intestinal tract. The round worms include the *Ascaris lumbricoides*, which resembles in shape a common earth-worm, the *Oxyuris vermicularis*, very small and thin, like a piece of cotton thread, about half an inch long; and the *Trichocephalus dispar*, which is readily recognized by its anterior end being thin and thread-like, while the posterior is much thicker. It measures from one to two inches in length. The three common varieties of tape worms are *Tænia solium*, *Tænia mediocanellata*, *Bothriocephalus latus*. The latter may be distinguished from the two first by the fact that the sexual openings occur in the middle of the segments, while in the others they are placed at the border. The *Tænia solium* differs from the *Tænia mediocanellata* in many particulars, the most obvious being its possession of from twenty-six to thirty hooklets on the head, which are absent in the *Tænia mediocanellata*. *Echinococcus* cysts are likewise sometimes found in the stools. Abnormal quantities of mucus and bile may be seen; and fat in large amount, when not accounted for by diet, usually indicates affection of liver or pancreas. To the presence of blood we have already alluded.

Microscopic Examination of the Fæces.—Fragments of food, including muscular fibre, connective tissue, fat cells and crystals, coagulated albumen, vegetable cells, etc., are readily to be detected in the stools. Besides these, we have to recognize certain elements which are derived from the tissues themselves, as for example, epithelial cells, mucous and pus corpuscles. blood corpuscles and crystals of various forms—triple-phosphate, cholesterin, Charcot's crystals, hæmic crystals, and balls of leucin. In the stools there also may be found many vegetable and animal parasites, the former including *Bacterium termo*, *Sarcina ventriculi*, and similar forms, while to the latter belong *Amœba coli*, various infusoria, and the eggs of the parasitic worms already described.

CHAPTER III.

Examination of the Abdomen.

The physical examination of the abdomen can only be made with advantage when the patient is in the recumbent

posture. It is well to have the shoulders slightly elevated by means of a pillow, so as to relax the abdominal muscles, which can be still further effected by causing the knees to be raised. The examination is conducted by means of inspection, palpation, percussion, and auscultation, each of which will be considered in turn.

Inspection of the Abdomen.

1. General Prominence.
   - (*a.*) Ascites.
   - (*b.*) Meteorism.
2. General Retraction.
   - (*a.*) Inanition.
   - (*b.*) Wasting Diseases.
   - (*c.*) Tubercular Meningitis.
3. Local Tumefaction of Various Organs.
4. Abdominal Movements.
   - (*a.*) Respiratory Movements.
   - (*b.*) Pulsatory Movements.
   - (*c.*) Peristaltic Movements.

The form of the abdomen varies greatly within physiological limits. A full dietary and a corpulent habit cause prominence in this region, whilst in old age and after prolonged inanition the belly sinks in, and its bony walls become unduly prominent. The enlargement of the abdomen in pregnancy is also physiological.

The pathological changes which inspection indicates may be considered under three heads—(1) General prominence, (2) General retraction, (3) Local tumefaction.

General prominence of the abdomen is found—

1. In *ascites*, where an effusion of fluid has taken place into the peritoneal sac. The fluid gravitates to the most dependent position; and, consequently, when the patient lies on his back, the anterior part of the abdomen is flattened, while the sides bulge, whereas, if the erect position be assumed, the prominence is greatest in the hypogastric region.

2. In *meteorism*, or accumulation of gas in the intestines. In this case a change in position does not affect the form of the abdomen, which is more spherical than in the former condition.

When the abdomen is greatly distended, from whatever

cause, the diaphragm becomes raised, the ribs pressed outwards, and the position and character of the apex-beat of the heart altered. The abdominal walls become smooth and glistening, the recti muscles are pushed asunder, and in the interval between them the peristaltic motion of the intestines may occasionally be observed. The umbilicus rises, first of all, to a level with the adjoining skin, and subsequently protrudes beyond it. The pressure exerted on the inferior vena cava gives rise to the development of the collateral venous circulation as a delicate blue network over the abdominal parieties, whilst obstruction to the portal circulation occasions distension of the veins at the umbilicus—this varicose condition being the more observable by reason of the prominence of the navel.

Retraction of the abdominal walls is met with in cases of inanition from whatever cause (particularly in œsophageal obstruction), and in all wasting diseases. It is also seen in various diseases affecting the nerve centres (tubercular meningitis), and is then attributed to contraction of the intestines, determined by the irritation at the base of the brain.

The bony walls become prominent, and the vertebral column, with the pulsations of the aorta lying on its left side, may be seen. The relaxed abdominal walls form pendulous folds, between which friction may produce ulceration of the skin.

Local tumefaction may occur in connection with various abdominal organs as follows:

Stomach.—Dilatation of this viscus gives rise to an oval swelling occupying the epigastrium, and extending chiefly towards the left. The position of the greater curvature may be indicated by a diagonal furrow running from the right downwards and to the left.

The outline of the stomach is best made out when it is artificially distended, according to the method originally recommended by Frerichs. The patient is made to swallow successively a solution of tartaric acid, and one of bicarbonate of soda, of each salt as much as may be carried on the point of a table-knife. The result of the mixture of these solutions in the stomach is the development in that viscus of a large quantity of carbonic acid gas, and consequent distension.* In a few seconds the position of the

* This method is not dangerous to the patient, provided that cases of gastric ulcer be excluded. The worst symptoms which an overdose

greater curvature, and the general outline of the stomach, can be made out with great distinctness. When the small intestine and colon become rapidly filled with the liberated gas, we may with safety diagnose incompetence of the pylorus, resulting from ulceration or carcinoma, or perhaps occasionally from abnormal innervation.

Tumors of the stomach sometimes cause visible prominence of the abdominal wall. They do not alter their position with the respiratory movements, and may thus be readily distinguished from tumors of the liver and spleen, which rise and fall with the respiration.

Liver.—In the healthy adult* the liver gives rise to no visible prominence of the abdominal walls. When enlargement takes place, it usually shows itself first under the margin of the ribs, in the right hypochondrium, unless the left lobe be chiefly affected, when the tumefaction appears in the epigastrium. The edge of the liver, or the tumor arising from that organ, when visible, may be seen to rise and fall with the respiratory movements—an important diagnostic point.

Splenic tumors, when of large size, cause visible tumefaction of the abdomen generally, particularly on the left side.

Tumors of the *kidneys and omentum* do not, as a rule, cause visible swelling.

Ovarian tumors are originally lateral in position, although they may develop to so great a size as to distend the whole abdomen.

Abdominal movements other than those of normal respiration may be seen—(*a*) respiratory, (*b*) pulsatory, (*c*) peristaltic.

The *respiratory movements* affect the position of all tumors which are attached to the liver or diaphragm. Splenic tumors are also similarly influenced, but to a less degree. Tumors of the stomach and pancreas, etc., are not so affected, unless they have formed adhesions to the diaphragm.

Pulsations of various kinds are met with in the abdomen,

---

might occasion would be vomiting, dyspnœa, and slight cyanosis—all disappearing in a few moments.

* In children the liver is normally large in proportion to the size of other viscera, and occasions a considerable degree of fulness of the abdomen, extending from the lower border of the ribs on the right side to the level of the umbilicus.

but their nature will be more conveniently considered under the circulatory system.

Peristaltic motions of the intestines may be observed in persons in whom the abdominal walls are abnormally thin (as in ascites, *vide antea*), or where such vermicular movements are unusually energetic (intestinal obstruction, irritation, etc.).

CHAPTER IV.

Palpation of the Abdomen.

At no portion of the body is the skilful application of the hand of more essential service to diagnosis than over the abdomen. By carefully pressing with the hand (which should be warmed) at various points with a kind of gentle kneading motion, we obtain by the sense of touch information regarding, 1*st*, the condition of the abdominal wall; 2*d*, the size, form, consistence, and mobility of certain of the abdominal organs, and whether any tumor be present within the cavity of the abdomen; and 3*d*, if there be general tumefaction of the abdomen, whether such distension is the result of accumulation of gas in the intestines (tympanites), or is due to the presence of serous or inflammatory exudation; and, in the latter case, whether such exudation be in the peritoneal cavity or be enclosed in a cyst of some kind or another.

The position of the patient is of the greatest importance. He must lie on his back, with head and neck slightly raised. and with the knees flexed and drawn up towards the abdomen. In most cases it is well to engage the patient in conversation while palpating the abdomen, as otherwise the abdominal muscles are usually involuntarily contracted, and the glottis closed. The air in lungs, retained there by the closure of the glottis, supplies the necessary resistance to this contraction of the abdominal muscles, and if this resistance be removed (as is best done by forcing the patient to open his glottis in speaking) the muscles have nothing to contract upon, and consequently become flaccid. The relaxation of these muscles may be still further aided by diverting the patient's attention, and should there be neces-

sity for it, the exhibition of chloroform will allow of a very perfect exploration of the abdominal cavity.

Abdominal Walls.—The temperature of the skin, the amount of subcutaneous fat, the presence or absence of œdematous or ephysematous swelling of the subcutaneous cellular tissue (see Chap. I.) are readily recognized by the palpating hand, and require no special mention here. Localized swellings of the abdominal wall, due to the presence of tumors, of inflammation or of abscess, may be mistaken for more serious affection of the abdominal organs themselves. The immobility of such swellings, their position being unaltered by the respiratory movements, or by a change in the position of the patient, will generally suffice to distinguish them. In reality, the physician has seldom any difficulty in satisfying himself of the seat of the swelling, whether in the parietes, or in the cavity of the abdomen. In difficult cases, such, for example, as when a deep-seated abscess over the liver simulates a hepatic abscess opening outwards, the history of the case, and the other signs and symptoms, suffice as a general rule to indicate the real seat of the abscess.

The abdominal muscles, and more especially the "recti," present, when contracted, certain inequalities in thickness which are occasionally mistaken by the inexperienced for abdominal tumors.

The various hernial protrusions which are found in the umbilical, femoral, and inguinal regions, belong more especially to the domain ot surgery.

Peritoneal Cavity.—Acute general peritonitis gives rise to great pain and tenderness on pressure over the whole surface of the abdomen. In chronic peritonitis a characteristic doughy resistance is usually felt over the affected part, accompanied with some tenderness on pressure. Transudation of fluid into the peritoneal cavity (ascites) gives rise to a feeling of fluctuation. This is best appreciated by placing the hand on one side of the abdomen, and giving a smart tap on the surface at a point diametrically opposite. The impulse of the wave so formed can usually be clearly felt when it reaches the opposite wall. If, however, the amount of fluid be small, no such undulation will be observed in the ordinary position. The patient may then be placed on his elbows and knees, when the fluid will gravi-

tate to the anterior part of the sac, and fluctuation can there be obtained.

Friction vibration can also occasionally be felt between two roughened peritoneal surfaces. It may be synchronous with the respiratory movements, and this most frequently if the visceral and parietal layers over the liver and spleen be the seat of the roughness (particularly in carcinoma of the liver). Friction vibration can also be induced in such cases by moving the peritoneal surfaces against one another, and pressure will at all times increase the strength of the friction.

Liver.—In the healthy adult, as a rule, only the left lobe of the liver can be felt by the palpating hand, giving rise to a slight feeling of resistance in the epigastric region. On very deep inspiration, however, the edge of the right lobe may sometimes be made to project so far beyond the costal margin as to offer appreciable resistance to the fingers. In children the liver is of such size as to be readily examined by palpation.

Either as a result of enlargement or of lowered position (due, for example, to the downward pressure of a pleural effusion), the liver may come within reach of the palpating hand, and then we have to examine the condition of its surface, the consistence of the organ, its size and general shape.

The surface of the liver may be smooth or rough. In amyloid and fatty degeneration, and in congestion, the surface of the swollen organ is smooth, a condition which is very readily recognized by palpation. In the case of cirrhosis, the uneven granular surface gives rise to a characteristic feeling of roughness when the abdominal wall is made to glide backwards and forwards over the surface of the liver. More marked irregularities of surface are found in carcinoma, the distinct nodules of which can be felt, and occasionally the umbilications which these nodules present.

Tenderness on pressure is met with in congestion and in all inflammatory affections of the liver, such as hepatic abscess, cirrhosis, catarrh of the bile ducts. In carcinoma it is often a very marked feature, although even in this affection it may be absent. There is usually no tenderness in the case of the waxy and the fatty liver.

Consistence of the liver is somewhat increased in fatty degeneration, still more so in congestion, and to a very marked

degree in waxy disease, when the lower edge may assume an almost knife-like sharpness. The presence of fluctuation will usually suffice to distinguish a hydatid tumor or an abscess from a solid growth.*

The *size* of the liver varies greatly. In some cases, as in acute yellow atrophy, the organ recedes so far into the concavity of the diaphragm as to be out of reach of palpation. In other instances (congestion, waxy degeneration, etc.) the lower edge may be found as low as the symphysis pubis. It must be carefully borne in mind that the position of the lower border is no safe guide to the size of the liver unless it be taken along with the position of the upper margin as ascertained by percussion.

Abnormalities in Shape.—The practice of tight-lacing not only forces the liver downwards, but also frequently so compresses the hepatic substance as to give rise to a deep furrow marking off the lower portion of the right lobe. This furrow can be readily detected by palpation. Still more obviously is the shape altered by the presence of a large tumor, cancerous or hydatid, growing from some particular part of the organ. It is most important to remember that hepatic tumors rise and fall with the respiratory movements, which is not the case with growths in the stomach, omentum, pancreas, colon, or kidney, unless they have become adherent to the liver.

Occasionally the gall bladder may be felt as a small pear-shaped tumor projecting from beneath the lower edge of the liver. Pressure, by emptying it of bile, may cause it to disappear, and in rare cases the presence of gall-stones in the bladder may be ascertained by palpation.

Spleen.—In the normal condition the spleen cannot be felt, and this is partly due to its deep-seated position, and partly to the fact that the splenic tissue is too soft to offer resistance to the palpating fingers. When, however, it becomes so enlarged as to reach the extremity of the eleventh rib, or to pass beyond it, then the spleen can be readily recognized. Increase in size of the spleen takes place in numerous diseases, such as leucocythæmia, amyloid dis-

* When a hydatid tumor lying near the surface of the liver is percussed, a peculiar and very characteristic tremor (hydatid fremitus) may be felt over it, due to the reflection, from side to side of the sac, of the undulations into which the fluid has been thrown.

ease, recent syphilis, intermittent fever, typhus, enteric, and scarlet fevers, etc.; and in addition, all diseases which produce obstruction to the portal circulation, directly or indirectly (such as cirrhosis of the liver and heart disease), cause splenic congestion, and, consequently, enlargement of that organ.

When the spleen is but slightly enlarged it can be best felt by tilting it upwards from the lumbar region on to the palpating hand. A feeling of increased resistance may thus be appreciated, although the limits of the organ may not be felt with any distinctness.

As the organ increases in size it projects from beneath the margins of the ribs towards the umbilicus, and rises and falls to a slight extent with the respiratory movements. The enlargement is proportionately the same in all diameters, and so the spleen retains its original shape. The splenic notch is readily felt, and is important as a certain indication that the tumor with which we have to deal is splenic. In leucocythæmia the spleen may attain an enormous size, and fill up the greater part of the abdominal cavity. Except in very rare cases (hydatid disease and carcinoma of the spleen) the surface of the swollen organ is smooth, and there is rarely any tenderness on pressure.

The consistency of the spleen is greatly increased in amyloid disease and in leucocythæmia. In congestive enlargement it is not so resistant, and in acute diseases the tumefied gland is of a very soft consistence. During the exacerbations of intermittent fever the spleen undergoes perceptible enlargement, while in cases of splenic congestion from portal obstruction, loss of blood from the stomach or intestines causes diminution in its size.

Pancreas.—Tumors of the head of the *pancreas* are rarely met with, and are difficult of diagnosis, owing to the way in which the gland is covered by the coils of the intestines, and to some extent by the lower edge of the liver. Hardness in such cases can usually be felt to the right of the middle line above the level of the umbilicus. It is deeply seated, not freely movable, and unaffected by the respiratory movements. The disease (which is almost invariably carcinoma) is seldom limited to the pancreas, but attacks the retro-peritoneal lymphatic glands and other neighboring parts.

Stomach and Intestines.—Tumors of the stomach (usu-

ally carcinomatous) are most common at the pylorus. They are readily felt as irregular, nodulated masses in the umbilical region, freely movable for the most part, and but little affected in position by the rise and fall of the diaphragm. When the greater curvature is the part affected, the tumour mass is found somewhat lower down, and to the left. Tumors of the lesser curvature and cardiac end of the stomach are rarely felt during life, as they lie so deeply in the concavity of the diaphragm.

Pressure over the stomach occasions pain in many diseased conditions of that viscus. It is most circumscribed in cases of gastric ulcer, and is often of very great severity.

In the intestines the retention of fæces, chiefly in the large intestine, may give rise to localized swelling at various points. These nodular masses are of a doughy consistency, and to a large extent disappear after purgation. Catarrhal and inflammatory conditions of the colon are apt to give rise to inflammation in the neighboring tissues, resulting in a swelling which is usually ill-defined, doughy, hard, and very tender on pressure. Such inflammatory processes usually take place round the cæcum (perityphlitis). Cancerous masses may occasionally be felt at various parts of the colon: the cæcum and sigmoid flexure being most commonly the seat of the disease. Peristaltic movements of the intestines are occasionally to be felt when the abdominal walls are thin or the movements very energetic, as in stenosis of the bowel.

Tumors of the *omentum* are rare. They are of very various nature: cancerous, tubercular, hydatid, etc., and when developed are readily felt through the abdominal wall. When affected with carcinomatous disease the omentum becomes thickened and retracted, and its lower hardened edge may occasionally be felt crossing the abdominal cavity.

The **Mesenteric Glands** are frequently the seat of tumors. They may be simply enlarged, along with other similar glands throughout the body, or they may be affected with cancerous, tubercular, or other deposits. They are smooth, hard, movable tumors of regular form. Occasionally they become fused together, along with other neighboring structures (loops of small intestines, retroperitoneal glands, etc.), into masses of considerable size, which, overlying the aorta, may have imparted to them a pulsatile movement.

The **Kidneys** are not, in their normal condition, within the range of palpation; but when they leave their position, or when they increase greatly in size, they may be felt. A floating kidney—that is, one the attachments of which are so loose as to allow of its moving more or less freely through the abdominal cavity—is recognized by its possessing the size and the characteristic renal shape, as well as by its great mobility, and by the presence of the pulsating renal artery, which may sometimes be felt entering at the hilus. The diagnosis is rendered more certain by the aid of percussion, as will be noticed under that head (*vide* p. 58).

Enlargement of the kidney occurs in hydronephrosis, echinococcus, carcinoma, etc., and the tumor is smooth or nodular according to its nature. It is recognized by its position and relations, its immobility, and its cylindrical shape from above, downwards. Inflammatory thickening round the kidney and perinephritic abscess may also be recognized by palpation.

The *urinary bladder*, when distended, forms a pyriform tumor above the pubes, and may reach the level of the umbilicus, or even, in extreme cases, to a still higher point.

Ovarian tumors in most cases are cystic, and may be so large as to distend the whole abdomen. When small in size they are usually on one side only, and gradually cross the middle line to assume an apparently central position. Fluctuation is generally easily made out. The characteristics of such growths, as well as of tumors of the *uterus*, physiological and pathological, will be considered in connection with the reproductive system.

Aneurism of the abdominal aorta may affect that vessel in any part of its course. If it lie very deep in the concavity of the diaphragm, it may not be capable of being felt with any distinctness, but where it can be reached a pulsating tumor is readily recognized. The pulsation must be distinguished from that produced by a tumor lying on the vessel, which is to be done by noting its true expansile character when compressed laterally between the fingers. Aneurisms of the main branches of the abdominal aorta also occasionally occur.

CHAPTER V.

Percussion of the Abdomen.

The theory of percussion will be considered hereafter, and need not detain us here. Its use in connection with the abdomen is to define the outline of organs which it is not possible to reach by means of palpation. As a rule it is best to use the forefinger of the left hand as a plexi-meter, laying it upon the surface of the abdomen, and eliciting a note by striking with one or more fingers of the right hand. The change of note as one passes off such a solid organ as the liver on to air-containing viscera, is usually sufficiently obvious.

Percussion gives information concerning—

1. The Condition of the Peritoneal Sac.
2. The Outline of the Liver.
3. The Outline of the Spleen.
4. The Outline of the Kidneys.
5. The Condition of the Stomach and Intestines.

Condition of the Peritoneal Sac.—When transudation of fluid takes place into the peritoneal cavity, it does not in the first instance affect the percussion note over the surface of the abdomen, since the small quantity of serum, which at first collects, gravitates towards the lowest portion of the sac; and whether this point lie in the pelvis or towards the spinal column (determined by the position of the patient, erect or supine), the collection is too far removed from the surfaces ordinarily subjected to percussion to allow of its influencing the note obtained.

As the quantity increases it gradually makes its presence manifest, causing a dull note to be heard on percussion over the lower parts of the peritoneal cavity. With further increase in the quantity of fluid, the dulness extends its area, until in extreme cases where the sac is greatly distended with transudation and the bowels compressed, the note over the whole surface of the abdomen becomes absolutely dull. In cases of medium severity, when the patient lies on his back, the fluid gravitates towards the lumbar regions, and the intestines float on its surface, so that the percussion note over the anterior surface of the abdomen

is clear and tympanitic, expressing the presence of large air cavities beneath (bowels), while on either side, as we pass towards the lateral and posterior regions, there is dulness corresponding to the position of the ascitic fluid. If the patient lie on his left side the right side will be the point towards which the air-containing intestines will float, whereas the fluid in the peritoneal cavity will gravitate towards the left. The change in the percussion note thus caused by alteration in the position of the patient is an indication of the presence of free fluid in the peritoneum, and is the more important seeing that it does not occur in the case of ovarian dropsy.

Ascites is either the result of increase in the blood pressure within the portal vein due to cirrhosis of the liver, tuberculosis of the peritoneum, etc., or is merely a part of the general dropsical condition caused by cardiac or renal disease.

Percussion of the Liver.—A considerable portion of the anterior and upper surface of the liver lies in contact with the anterior wall of the abdomen, and consequently over this area the percussion note is more or less absolutely dull expressing the presence of a solid organ underneath. This area is spoken of as the *absolute hepatic dulness.*

Above this the liver recedes from the chest-wall and becomes separated therefrom by a layer of lung of gradually-increasing depth. In percussion from above downwards in the right parasternal line, the level at which the percussion note indicates that the subjacent air space is being encroached upon and rendered shallower, corresponds (in the normal condition) to the highest point to which the liver reaches under the diaphragm, and at this level the deep or *relative hepatic dulness* commences.

The tympanitic resonance of the neighboring abdominal organs which contain air enables us to mark out with considerable ease the lower border of hepatic dulness. The thinness of the lower edge of the liver necessitates that percussion should be made very lightly in order that we may avoid, as much as possible, the transmission of the vibrations to parts in the vicinity. It is best practised, in this particular instance, by tapping very gently with the fore-finger on an ivory pleximeter.

The *lower border of the liver* begins at the left, close to the apex of the heart, and passes diagonally downwards and to-

wards the right, crossing the middle line at a point midway between the ensiform cartilage and the umbilicus, and joining the margin of the ribs at an acute angle in the mammary line. From this point backwards to the axillary line the lower border corresponds pretty closely with the margin of the ribs. In many cases careful percussion will detect the presence of the gall-bladder, as a small rounded tumor projecting downwards from the edge of the liver.

The upper border of absolute hepatic dulness corresponds to the lower edge of the right lung, except in regard to the left lobe, where it passes imperceptibly into the cardiac dulness. At the right border of the sternum it lies at the level of the 6th rib; in the mammillary line it corresponds to the upper border of the 7th rib; and in the axillary and scapular lines it reaches respectively the 8th and 9th ribs.

The upper limit of the deep or *relative hepatic dulness* lies about three inches above the absolute dulness. In the mammillary line the percussion note can usually be noticed to become dull about the 4th intercostal space, or 5th rib.

The movements of respiration change the position of the liver. Deep inspiration depresses the lower edge considerably, while full expiration permits of a corresponding elevation. But in addition to such alterations in the level at which the lower border of the liver stands, the respiratory movements affect the position of the upper border of the absolute dulness to a much greater extent. This latter alteration in hepatic dulness does not express so much an alteration in the position of the liver, as the rise and fall of the lower border of the right lung, and the extent of the complimentary pleural space occupied by pulmonic tissue.

Changes in the position of the body also cause slight differences in the position of the liver, the organ gravitating towards the most dependent side.

The hepatic dulness may be greatly altered without any actual change in the size of the liver. Thus the colon, distended by air or by a coil of the small intestine, may be forced upwards between the surface of the liver and the abdominal parietes, thus preventing the true lower border from being found by percussion, and leading to an apparent diminution in the size of the liver. In pulmonary emphysema the right lung extends lower down than normally, and the upper border of absolute liver dulness is thus depressed. Again, the liver may be elevated abnormally within the concavity of the diaphragm, by reason

of increased intra-abdominal pressure; a greater proportion of the organ will be thus overlapped by lung, and the absolute dulness diminished. If the two last conditions coexist, all trace of absolute hepatic dulness may fail, and this may occur when the liver is of normal size.

From all this it follows that the extent of the absolute hepatic dulness is no safe guide to the size of this organ, and in all cases it is best to measure from the upper level of the deep or relative dulness to the lower border of the liver. Unfortunately, in some cases such measurements cannot be accurately made (right pleural effusion, pronounced ascites, etc.), but when percussion can determine the deep dulness, the position of the lower border can in such cases usually be ascertained by palpation.

Displacement of the liver takes place more frequently downwards than upwards. The causes of displacement *downwards* are—(1) Emphysema of the lungs, when both hepatic lobes are equally depressed; (2) Pleuritic effusion on the right side, which causes depression of the right lobe of the liver, with perhaps slight elevation of the left lobe; (3) Right pneumothorax producing the same conditions. To these may be added, as much rarer causes of downward displacement, various tumors of the mediastinum and of the diaphragm, and encapsuled peritoneal effusions between the diaphragm and the upper surface of the liver. The left lobe of the liver may be slightly depressed by large pericardial effusions, and by effusion of liquid or gas into the left pleural cavity.

Displacement upwards occurs less frequently, and to a less extent, than depression. It is occasioned by any condition which produces an increased pressure in the abdominal cavity—ascites, meteorism, ovarian cysts, etc.—and possibly also by cirrhosis of the right lung.

Hepatic Enlargement.—The increase in size of the liver may be very marked. It may in rare cases rise as high as the 2d rib (Gerhardt), while its lower edge may reach to a point close to the symphysis pubis. The chief causes of great enlargement of this organ are hydatid tumors, carcinoma, and waxy disease. It is found to a less degree in hepatic congestion (as in mitral disease), occlusion of the bile ducts, fatty degeneration, and in certain cases of cirrhosis. (The alteration in the shape of the liver, caused by the practice of "tight-lacing," may simulate actual enlargement.)

Diminution in the size of the liver occurs in the later stage of cirrhosis, and in acute yellow atrophy of the liver. The organ, as it becomes smaller, leaves the surface of the abdomen and retreats into the concavity of the diaphragm. Its place is occupied by small and large intestines, and, in consequence, all trace of hepatic dulness may disappear. This extreme diminution is met with in the latter disease, whilst in cirrhosis, when the liver is much contracted, the percussion of the lower border is prevented by the almost invariable presence of ascites at that stage.

Percussion of the Spleen.—Placed obliquely in the cavity of the abdomen, the spleen lies with its upper and posterior extremity opposite the tenth dorsal vertebra in the concavity of the diaphragm and somewhat overlapped by the left lung. From this point the gland passes downwards and forwards to terminate slightly behind the extremity of the eleventh rib. Its upper and anterior border runs parallel with the ninth rib, while the posterior and lower border follows pretty closely the eleventh rib.

The percussion of the spleen is peculiarly important, because when the organ is but slightly enlarged, it is practically inaccessible to palpation. In consequence of the nearness of large air-cavities in the stomach and colon, it is necessary to percuss very lightly, in order to obtain the splenic dulness, and not allow the note to become obscured by reason of the tympanitic resonance of these organs.

The spleen is of variable size, and as age advances it atrophies so that a small area of dulness may be met with under physiological conditions.

Respiratory movements affect the position of the splenic dulness, deep inspiration depressing it and diminishing its size. When the patient lies on the right side the spleen tends to gravitate towards that direction, and the splenic dulness diminishes or disappears. When the spleen is not greatly enlarged, it is best to percuss it when the patient is in the erect position, with the left arm removed from the the side.

The condition of the stomach has an important influence on the percussion of the spleen. If the fundus is greatly distended with food, it occasions a dull note on percussion in the neighborhood of the spleen in such a way as altogether to prevent the differentiation of the splenic dulness. On the other hand, if the stomach be distended with gas to

a marked degree, it becomes difficult to determine with any exactness the limits of the splenic dulness, because of the tympanitic resonance of the gastric cavity, which even a very light stroke can hardly fail to produce.

Conditions similar to those which cause upward displacement of the liver (p. 56) force the spleen in the same direction under the lower margin of the left lung; and in this case, as well as when the spleen is overlapped by emphysematous pulmonary tissue, all traces of splenic dulness disappear.

The presence of ascitic fluid round the spleen will prevent its limits from being determined by percussion, and meteoric distension of the intestine with gas will cause a diminution in the size of the splenic dulness.

In pleuritic effusion and pneumothorax on the left side, as well as in pulmonary emphysema, the spleen is depressed, but in none of these conditions is it possible to define its limits by percussion.

Enlargement of the spleen is readily made out by means of percussion; but this method does not give any clue to the cause of the tumefaction, as in all cases the gland enlarges uniformly in all directions. When it passes beyond the borders of the ribs, it is best recognized by means of palpation. The various forms of splenic tumor have been already referred to and need not now be recapitulated.

Percussion of the Kidneys.—It is only in the rarest of cases that such a method of examination need be resorted to to aid in forming a diagnosis, and this because (1) the most frequently occuring renal diseases are not accompanied by so great an amount of alteration in the dimensions of these organs as to be appreciable by percussion; and (2) those cases of renal tumors where the tumor mass might be thus recognized are capable of far earlier and much more thorough investigation by palpation. Renal percussion may in fact be regarded as of no importance, except perhaps in the single instance of the diagnosis of floating kidney, where the absence of the normal area of renal dulness on one side might confirm the opinion. But as undoubtedly in many cases the percussion of the kidneys *can* be carried out, the subject must not be entirely ignored here.

The percussion stroke must be very firm, and is best

given by means of a hammer and an ivory pleximeter. In persons in whom there is great development of the subcutaneous adipose tissue, the kidneys cannot be percussed.

The upper border of the renal dulness cannot be defined, as it passes imperceptibly into that of the liver or spleen, and the internal border lying next the spinal column is also incapable of definition. It is then chiefly the outer border, lying in the lumbar region, parallel to and about three finger-breadths distance from the spinal column, that can be marked out, while in some few cases the position of the lower border, close to the crest of the ilium, can also be ascertained.

The patient must be laid prone, the anterior surface of the abdomen being supported on cushions. The lower edges of the hepatic and splenic dulness in the scapular line on either side must first be marked out. Immediately below these levels the tympanitic note of the colon or of the stomach can be heard. If now we percuss inwards on either side towards the vertebral column, the renal dulness will be reached at the distance already indicated. The length of the renal dulness is usually from three to four inches, and occasionally, as I have already said, the lower border may also be defined by percussion.

[The *urinary bladder* does not enter the abdomen unless distended, and then it can readily be percussed out as a pear-shaped tumor lying in the middle line, and giving a dull note.]

Percussion of the Stomach.—When the stomach is filled with food it is impossible to define its boundaries in any way by means of percussion; but when the cavity of the viscus is moderately distended with air it gives on percussion a tympanitic note of so low a pitch and of so long duration that it is readily distinguishable from the tympanitic notes obtained from the neighboring hollow viscera, and this by reason of the greater size of the air-cavity. Should, however, the distention of the stomach with gas increase beyond a certain point, the walls are also thrown into vibration, and a metallic ring results, which renders the definition of the gastric outlines more difficult.*

The position of the stomach is such that its border can

* I have already alluded to the method of artificially distending the stomach with gas for purposes of diagnosis.

be satisfactorily defined only in one direction, *i.e.*, in the line of the greater curvature; but the ascertaining of the position of this border is sufficient to enable us to say whether the viscus be enlarged or not.

It is best, first of all, to mark out the borders of the various other organs which surround the stomach, namely, the liver and spleen, as well as the position of the diaphragm. It is then found that the deep-pitched note of the stomach extends from the middle line to the left hypochondrium, neither passing to the right of the middle line nor below the level of the umbilicus. If either of those limits be overstepped at any point, the stomach is enlarged.

Over the *intestines* the percussion note is normally tympanitic, but is higher in pitch than over the stomach.

When the bowels become distended with liquid or solid contents this note ceases to be heard.

Great distention with gas allows the walls of the intestines to pass also into vibration, thereby producing a metallic note.

Auscultation of the Abdominal Organs.—Auscultation of the abdominal organs is rarely of value as an aid to diagnosis, if we except the auscultation of the uterus in pregnancy, which does not concern us here, and aneurisms of the abdominal aorta and its branches, which will be better considered in connection with the circulatory system.

CHAPTER VI.

Hæmopoietic System.

Lymphatic Vessels—Inflammation—Narrowing and Dilatation—Rupture.
Lymphatic Glands—Inflammatory Enlargement—Scrofula—Syphilis—Cancer—Adenia—Leucocythæmia—Plague—Glanders and Farcy.
Ductless Glands—Spleen—Thyroid—Thymus.
Examination of the Blood—Microscopic Examination—Enumeration of Corpuscles—Estimation of Hæmoglobin.

Under this heading we have to consider the lymphatic system, the various ductless glands (spleen, thyroid, etc.), and the microscopic characters of the blood.

1. **The Lymphatic Vessels**, as well their capillary network as their larger trunks, and the thoracic duct itself, undergo various pathological changes, which, although in great measure belonging to the domain of surgery, come frequently under the observation of the physician. For convenience of description the following order will be here followed:

(*a.*) *Inflammation* of the lymphatics (lymphangitis) depends almost invariably upon the absorption of some irritating (usually septic) material from the tissues. It sometimes follows some slight wound or sting, but often results from gonorrhœal, syphilitic, cancerous, or erysipelatous irritation. The superficial lymphatics show themselves as pale red lines passing up towards the glands, and the larger vessels may be felt as hard and knotted cords, and pressure over them is painful. There is usually some swelling of the surrounding tissues.

(*b.*) *Narrowing and Dilatation.*—Narrowing and obstruction of the lymphatic vessels may arise from the pressure of neighboring tumors, from inflammation, thrombosis, etc. Dilatation (Lymphangiectasis) is commonly the result of this narrowing, or obliteration, but it may arise also from disease of the lymphatic glands preventing the flow of lymph. Sometimes this dilatation exists without giving rise to any symptom, but when the lymphatics affected are superficial, they may be readily felt. This occurs most frequently on the inner sides of thighs, on the scrotum, penis, and lower surface of the abdomen. Nodular vesicles like sago grains, may then be felt lying under the skin, which sometimes spontaneously rupture, discharging lymph. They are to be distinguished from varicose veins by their position, form, and color. Sometimes the enlarged lymphatic trunks take the form of a tumor (lymphangioma), usually in the tongue, cheek, or neck.

The *Thoracic Duct* itself is sometimes narrowed, or occluded, most commonly as the result of the pressure of thoracic tumors (aneurisms, cancerous and tubercular disease of the mediastinal glands), and sometimes, perhaps, from inflammation of its coats, or thrombosis. This obstruction is followed by dilatation of the duct (which may also arise from stasis in the large veins at the root of the neck, the result of heart disease), and this dilatation, especially if it take place slowly, may give rise to no symptom. Very often, however, it is followed by marked wasting and

anæmia, and sometimes by ascites, and even hydrothorax, the fluid poured out in the pleural or peritoneal cavity resembling chyle.

(*c.*) *Rupture.*—The discharge of lymph (lymphorrhagia) follows every wound, and only becomes important when it flows from a large trunk, a condition which may be caused by wound, by ulceration, or by rupture following previous dilatation. Its diagnosis can present no difficulty when the effusion of lymph takes place externally. Rupture is most frequent in the inguinal region. The lacteals sometimes rupture (the result of congestion from the pressure of a tumor), discharging their chyle into the peritoneal cavity, and usually giving rise to no symptom. Several cases are on record of rupture of the thoracic duct, caused by wound, or by the bursting of a neighboring abscess. The symptoms are obscure, but the presence of rupture of the duct might be diagnosed by the flowing of chyle from the wound, or by the accumulation of that fluid in the pleural cavity.

Chylous urine, probably the result of rupture of the renal lymphatics, will be considered under the urinary system.

Neoplasms of the lymphatic vessels are so rare, and so obscure, as not to warrant their consideration in these pages.

2. **The Lymphatic Glands.**—Enlargement of the lymphatic glands may arise from various causes.

(*a.*) *Inflammatory.*—This may be the direct result of a blow, but more commonly arises from the absorption of irritating material from an abrasion. Such enlargement is common at the elbow, axilla, knee, and groin. The chain of femoral glands may enlarge as the result of a wound on the leg or foot, while irritation on the penis affects the inguinal chain (bubo). The glands at the back of the neck frequently swell where there exists any irritation of the scalp, and the cervical glands become enlarged in diphtheria and scarlatina.

(*b.*) *Scrofula.*—In strumous persons, particularly in childhood and early life, the glands of the neck enlarge, slowly suppurate, and caseate. In cases of tabes mesenterica scrofulous glands may be felt through the abdominal wall.

(*c.*) *Syphilis.*—The adenitis (almost invariably inguinal) which follows a soft chancre is called the "virulent bubo." It comes on, generally, about a week after infection, is accompanied with acute pain, and ends in suppuration. The "multiple bubo," which follows the hard chancre after an

interval of about three or four weeks, is, as a rule, painless; there is no suppuration, and the induration proceeds slowly from gland to gland in the inguinal chain until all are affected. They seldom exceed a walnut in size, are hard, and roll about freely under the finger. In the secondary stage of syphilis many of the lymph glands throughout the body are enlarged (particularly at the back of the neck and the inside of the elbow).

(*d.*) *Cancer.*—In the neighborhood of cancerous tumors the lymphatic glands become enlarged and hard, and constitute an important element in diagnosis and prognosis. The most common example of this is the enlargement of the glands in the axilla and above the clavicle, which follows carcinoma of the breast.

(*e.*) *Adenia, or Hodgkin's Disease.*—This affection is characterized by an enlargement of the lymphatic glands throughout the body. They may attain to great size, but do not suppurate. There is marked anæmia, a diminished number of red blood corpuscles, but no increase of the white, and the spleen is generally enlarged. The cervical glands are usually the first to be affected.

(*f.*) *Leucocythæmia.*—In some cases of this disease there is a general enlargement of the lymphatic glands throughout the body, along with that of the spleen. It is readily distinguished from adenia by the increase in the white blood corpuscles which is present.

(*g.*) In the *Plague*, and also in *Anthrax*, *Glanders*, and *Farcy*, acute glandular swellings form in the axilla, groin, and neck.

3. **The Ductless Glands.**—*The Spleen* has been already considered, under the abdominal system, and need not be again alluded to.

The Thyroid.—The very rare inflammation and simple enlargement of this gland, as well as the malignant and cystic growths which form in it, belong to the domain of surgery. There remain for consideration only two diseases:

(*a.*) *Bronchocele*, or *Gôitre*, is an endemic disease, prevailing chiefly at the base of high mountains. The thyroid is enlarged, the whole or part of the gland being affected, and presses upon neighboring parts, leading to difficulty of breathing and swallowing. Gôitre is often associated with mental deficiency (cretinism).

(*b.*) *Exophthalmic Gôitre—Graves's Disease.*—In this affec-

tion the swelling of the thyroid (usually the right lobe) is of a vascular character—soft, elastic, and pulsating. It is accompanied with marked pulsation in the vessels of the head and neck, palpitation of the heart, prominence of the eyeballs, and other nervous symptoms, and is most common in females below middle age.

Thymus.—Diseases of the thymus hardly come within the limits of possible diagnosis, except those lymphatic and malignant tumors of the anterior mediastium, which have their starting-point in that gland.

BLOOD.

The examination of the blood has of late years attained to such importance as an aid to diagnosis, and as a guide in the subsequent treatment of many diseases, that the various processes employed demand a somewhat minute description. Limiting ourselves to those methods which may be truly called *clinical—i.e.*, which can be carried out at the bedside of the patient—and therefore excluding the minute chemical analysis which can only be performed in the laboratory, and for which the comparative infrequency of blood-letting gives but few opportunities during the life-time of the patient, we shall find the lines of investigation to be mainly three—

1. Microscopic examination of the blood.
2. Enumeration of corpuscles it contains.
3. Estimation of the hæmoglobin.

1. **Microscopic Examination of Blood.**—To obtain a drop of blood for examination, the end of the patient's finger must be carefully cleansed, and a prick made with a needle. The finger must not be squeezed or compressed in any way, as this tends to alter the proportion of serum and corpuscles which the blood contains. The drop of blood so obtained should be received on a clean microscope slide, the cover-glass applied, and the microscopic examination made as speedily as possible. The pathological changes usually met with may be divided into three classes.

(*a.*) Alterations in the form of the normal constituents of the blood.

(*b.*) Variations in their number.

(*c.*) The presence of foreign substances.

(*a.*) *Alterations in the form of the Normal Constituents of*

the Blood.—In anæmia, particularly in the pernicious variety of that disease, the red blood corpuscles are frequently found to be much diminished in size, and in many cases to have a pale appearance due to loss of hæmoglobin. In other instances they tend to assume curious and irregular shapes of various kinds. The changes in the colorless corpuscles are not so distinctive, but not unfrequently they may be found, in case of anæmia and leucocythæmia, to be abnormally refractive, and to contain granules resembling oil-globules. In various cachectic conditions the protoplasmic granules which are to be found occasionally in normal blood become much increased in number, and adhere together so as to form larger or smaller masses.

(*b.*) *Variations in the number of the Blood Corpuscles.*—Whilst in many cachectic conditions, as, for example, in phthisis, the number of leucocytes in the blood may be above the normal (leucocytosis), it is in leucocythæmia that this increase is by far most observable, and by means of the microscopic examination of the blood alone, a diagnosis may be arrived at. The number of leucocytes present in the field may be very great, even in pronounced cases surpassing that of the red corpuscles. In the lymphatic variety of the disease the size of the leucocytes is very much that of ordinary lymph corpuscles, and they contain as a rule but one nucleus, while in splenic leucocythæmia they are larger in size, and usually possess more than one nucleus. The diminution in the number of red corpuscles in anæmia, though not so striking as the increase of leucocytes just mentioned, is of considerable clinical interest.

(*c.*) *The presence of Foreign Substances.*—Among these may be mentioned, in the first place, the dark brown or black pigment granules, which are sometimes found in considerable numbers in the blood after malarious fever. This condition is spoken of as *melanæmia.*

More important is the presence of the various micro-organisms which appear in the blood in certain diseases. I do not think it necessary here to refer to more than one or two of these forms, those namely in which the diagnosis rests much or mainly upon the evidence supplied by the microscopic examination of the blood.

Bacillus Anthracis.—This form of organism, which is morphologically identical with the hay bacillus (*B. subtilis*), is found in the blood of persons suffering from the disease

which is known under the different names of anthrax, woolsorter's disease, splenic fever, and malignant pustule. The blood is found to be filled with short rods, somewhat exceeding in length the diameter of a red blood corpuscle, which, as the disease advances, become changed into long thread-like filaments. These forms are usually to be made out with very considerable ease, but they will be rendered more distinct if the blood be allowed to dry on the cover-glass, and be then stained with methyl violet, or other appropriate reagent, in the manner recommended by Koch.

Spirochæte Obermeierii.—This spirillum, which is found in the blood of patients suffering from recurrent fever, possesses a very characteristic appearance. On examining a specimen of such blood with the microscope, it will be seen that, moving actively among the blood corpuscles, are a number of thread-like organisms. Their length is usually three or four times greater than the diameter of a red corpuscle, and in shape they resemble a cork-screw. Their movements are sometimes so powerful as to move the blood corpuscles with which they may come in contact in their course.

Besides these there are, as I have said, a considerable number of micro-organisms met with in the blood of different diseases, but although their form is usually sufficiently distinctive, yet their recognition by means of the microscope cannot be held to have as yet assumed sufficient practical diagnostic importance to justify any detailed description in these pages.

Mention must, however, be made of a much more highly developed parasite which is occasionally met with.

Filaria sanguinis hominis is the name which Lewis has given to a nematode worm which is found in the blood of persons suffering from chyluria, certain forms of endemic hæmaturia, elephantiasis, lymphoid affections, etc. These filariæ are exceedingly minute, and are the sexually immature form of the *Filaria Bancrofti*, a long thread-like worm which has been found in lymphatic abscesses. It has been shown that mosquitos and other blood-sucking insects play an important part in the dissemination of the *Filaria sanguinis hominis.*

2. **Enumeration of the Corpuscles in the Blood.**—This method of investigation, which was first attempted by Vierordt, has been followed out by numerous observers,

each of whom has described a special form of apparatus for the purpose. While the most recent modifications of Malassez's instrument, or the apparatus of Abbé-Zeiss, probably give the most rigidly accurate results, I will here describe the instrument designed by Dr. Gowers,* which is exceedingly handy, and is the form most frequently used in this country.

Gowers's *Haemacytometer* consists of (1) a small pipette, which when filled to the mark on its stem contains 995 cubic millimetres; (2) a capillary tube marked to contain five cubic millimetres. Each of these is furnished with an india-rubber tube and mouthpiece; (3) a small glass jar in which the blood is to be diluted; (4) a glass stirrer for mixing the blood and the solution in the jar; and (5) a brass stage plate carrying a glass slip, on which is a cell $\frac{1}{5}$th of a millimetre deep. The bottom of this is divided by intersecting lines into $\frac{1}{10}$th millimetre squares. Upon the top of the cell rests the cover-glass, kept in position by means of two springs.

The diluting solution may vary. One which answers well consists of a solution of sulphate of soda in distilled water, having a specific gravity of 1025.

When an observation is to be made, 995 cubic millimetres of the solution are measured by means of the pipette, and placed in the mixing jar. The patient's finger is now to be pierced with the spear-pointed needle supplied along with the instrument, and a drop of blood obtained without any squeezing of the finger, which, as I have already said, alters the proportion of serum and corpuscles in the blood. Five cubic millimetres of this blood are drawn into the capillary tube, and then blown into the diluting solution in the jar, any superfluous blood being previously removed from the point of the pipette by means of a soft cloth. The two fluids are well mixed by rotating the stirrer between the finger and thumb, after which a small drop is placed in the centre of the cell, and the cover-glass applied. The drop must lie in the *middle* of the cell, and must not not touch its sides. The slip is now placed upon the stage of a microscope, and the lens focussed for the squares, which are marked out by lines drawn on the floor of the cell. In a few minutes the corpuscles sink through the drop and

* Made by Hawksley, 300 Oxford Street, London. A full description of the instrument will be found in *The Practitioner* for 1878.

come to rest on these squares. The number in ten squares is then counted, and this multiplied by 10,000 gives the number in a cubic millimetre of blood.*

The most recent calculations show the number given by Vierordt—5,000,000 per cubic millimetre—is approximately correct. The average number in two squares is 100, and Gowers proposes to take this quantity as the "hæmic unit." This is a very convenient way of calculating, for the number of corpuscles per "hæmic unit," *i.e.*, in two squares (counting ten or twenty squares, and taking the mean), expresses the percentage proportion of the corpuscles to that of health. The number of white corpuscles in ten or twenty squares is easily counted, and the proportion of white to red ascertained. The normal *maximum* of white per two squares (hæmic unit) is .3.

In all conditions of anæmia and cachexia the number of red corpuscles undergoes diminution, and by examining their number from time to time we can obtain most valuable and trustworthy indications regarding the progress of the malady and the effect of our treatment.

3. **Estimation of Hæmoglobin.**—Various instruments have been devised for this purpose by Malassez and others. That of Dr. Gowers,† which I prefer both on account of its simplicity and the accuracy of the results which it gives, consists of two glass tubes of the same diameter, one of which contains a standard color-solution ‡ (glycerine carefully tinted by means of carmine and picrocarminate of ammonia), while the other, in which the blood to be tested is to be diluted, is graduated so that 100 degrees = two cubic centimetres. There is also a capillary pipette graduated to hold twenty cubic millimetres, a bottle with a pipette-stopper to contain distilled water, and a guarded needle to prick the finger.

* Probably it would conduce to greater accuracy if the mixture of blood and solution were accomplished by means of Potain's "Malangeur," and if a special cover-glass ground absolutely level were supplied along with this apparatus; and, further, if this cover-glass were fixed in such a manner to the slip that it might be steadily lowered on to the drop by means of a rack movement. All of these arrangements have been adopted by Malassez in the newest model of his instrument.

† Described in full by Dr. Gowers in the *Transactions of the Clinical Society of London*; vol. xii., 1879.

‡ The tint of this standard solution corresponds exactly to that of a dilution of twenty cubic millimetres of blood with 1980 cubic millimetres of distilled water, *i.e.*, a dilution of one in a hundred.

The method of using this instrument is as follows. The two tubes having been placed upright in the small wooden stand supplied for the purpose, a few drops of distilled water are placed in the bottom of the graduated tube. The blood having been obtained from a prick in the manner already described, twenty cubic millimetres of the blood are measured off by means of the pipette, and injected into the distilled water in the graduated tube, which must then be quickly shaken to ensure thorough mixture. More distilled water must now be added drop by drop until the tint of the diluted blood is the same as that of the standard. The degree of dilution as indicated by the graduation expresses the amount of hæmoglobin as compared with that of the standard, and as this is a dilution of one hundred, the degrees of dilution required to obtain the same tint represent the percentage proportion of the hæmoglobin to that of normal blood.*

If the corpuscular richness of the blood is ascertained by means of the hæmacytometer, we are able to compare this with the amount of hæmoglobin in a very instructive manner. Thus, a fraction, of which the numerator is the percentage of hæmoglobin, and the denominator the percentage of corpuscles, will express the average value of each corpuscle.

CHAPTER VII.

Circulatory System.

SUBJECTIVE PHENOMENA.

Before addressing ourselves to the physical examination of the heart, there meet us for consideration certain symptoms of a more or less subjective kind.

1. *Pain.*—In anæmic persons, and particularly in women suffering from uterine disease, from chlorosis, or from nervous affections, pain over the region of the heart is fre-

---

* Thus if the tints are identical when the dilution has reached 80 degrees, the blood contains only 80 per cent of the normal quantity of hæmoglobin.

quently complained of, and true cardiac pain may likewise be simulated by neuralgia in the chest wall. In heart disease of any kind, and particularly in fatty degeneration associated with gout, pain may be a more or less prominent symptom. In its most pronounced form—angina pectoris—it comes on in recurring attacks of short duration, but of extreme severity. The first of the attacks usually occurs when the patient is making some exertion. The chest feels as if held in a vice, the pain, which is always severe, and which may be of the most intense character, radiates from the heart to the shoulders, and down the left arm, or down both arms to the wrist, breathing almost ceases, the countenance sometimes becomes livid, and consciousness may be lost. In other cases the attacks may be very frequent, several in a day, or even one after the slightest exertion of body or of mind. The attack passes off as rapidly as it came on, and the patient may be free from its repetition for months or years. The pain of aortic aneurism is more lancinating and more continuous than angina pectoris.

2. *Palpitation.*—The abnormal perception of excited pulsation in the heart or aorta is very frequently due to mental excitement, to dyspepsia, flatulence, anæmia, or nervous debility, and is also met with in cases of exophthalmic gôitre. It also occurs as a result of organic disease of the heart, and in such cases it will be found to be aggravated by exertion. Derangements of rhythm will be noticed hereafter.

3. *Fainting* (*syncope*), which is primarily due to failure of the heart's action, is usually ushered in by a train of symptoms of which the chief are—pallor of the face, chilliness, cold perspirations, a feeling of weakness, of sinking in the epigastrium, and of sickness, pulse small and rapid, or slow and irregular, dimness of vision, ringing in the ears, and gradually increasing unconsciousness. Syncope may be due to organic disease of the heart, to nervous disturbance of the cardiac action (central or reflex), to intense mental emotion (hysteria), to deficiency of the blood supply to the heart muscle, or to want of blood in its cavities.

CHAPTER VIII.

CIRCULATORY SYSTEM—(*continued*).

INSPECTION.

THE cardiac or *præcordial region* corresponds to the lower part of the anterior mediastinum. It may be said to extend vertically from the second interspace to the sixth cartilage, and transversely from the apex-beat to a point about three-quarters of an inch to the right of the sternum.

The region so marked out overlies the heart, and the margins of both lungs which overlap it. More deeply still lie the organs contained in the posterior mediastinum.

In this chapter will be considered (1) the form and appearance of the præcordia; and (2) the various pulsatory movements which show themselves on the walls of the thorax.

Præcordia.—A slight degree of bulging of the thoracic wall in the cardiac region is more readily detected by simple inspection than by means of measurement. It may be the result of curvature of the spinal column anteriorly and to the left, but is more commonly caused by cardiac hypertrophy, pericardial effusion, aneurismal and other tumors adjacent to the heart, or circumscribed pleuritic effusions. When effusion takes place into the pericardial sac, the intercostal spaces widen, they become raised to the level of the ribs, and ultimately may even protrude beyond them.

Depression of the præcordial region, on the other hand, may take place during the absorption of a pericardial effusion, and may remain permanently if adhesion has taken place between the visceral and parietal layers of the pericardial sac.

Bulgings caused by aneurisms lie almost without exception above the fourth rib.

Pulsations.

1. The *Cardiac Impulse.*—In health the apex-beat is found in the fifth interspace, about two inches from the left margin of the sternum, and its area does not exceed a square inch in extent. In childhood, however, and in persons who have a short and wide thorax, it may stand as high as the fourth interspace, and may be thrown somewhat farther to

the left; whilst in old age, and in persons whose thorax is very long and narrow, the cardiac impulse is depressed to the sixth interspace.

While natural breathing does not affect its position, deep inspiration and expiration cause respectively depression and elevation of the apex-beat.

When the patient lies on either side, the apex-beat is deflected in a corresponding direction. This alteration is more marked towards the left.

[Pathological changes in the position of the apex-beat will be considered under the head of Palpation.]

Systolic Indrawing of the thoracic wall is of two varieties. (1) A recession, which is exactly synchronous with each ventricular systole; and (2) an indrawing, which immediately succeeds the retirement of the apex of the heart from the chest wall.

The former variety (which is of little practical importance) is sometimes met with in healthy persons (particularly children), in whom the chest walls are unusually thin. It occurs in the third and fourth interspaces, and is simply the result of that recession of the base of the heart which is synchronous with the forward movement of the ventricles. The chest walls are sucked inwards (or rather forced inwards by atmospheric pressure) to prevent the formation of a vacuum behind them which would otherwise take place.

The second form is seen at the apex, aud is, according to Skoda, pathognomonic of adherent pericardium.* If the adhesion be extensive, not merely the intercostal space but even the ribs may be drawn inwards, following the apex of the heart.

2. *Pulsation at the Root of the Neck* may be arterial or venous.

Pulsation in the carotid arteries becomes evident whenever the heart's action is increased in strength (as after great bodily exertion, or from mental excitement), but in its most pronounced form such pulsation is seen in cases of hypertrophy of the left ventricle, along with aortic incompetence. Pulsation in the jugular fossa when well marked

---

* This sign is not, however, invariably present in such cases. and though adherent pericardium is by far its most common cause, yet it may occur in cases in which the normal movements of the heart are otherwise hindered (Friedreich).

usually points to simple or aneurismal dilatation of the aorta.

Swelling of the jugular veins is found in cases in which there is some obstruction to the return of blood to the heart, whether that obstacle be situated in the systemic or pulmonary circulation. If from any cause the right ventricle be unable to empty itself completely of blood, it becomes gorged, and, reacting on the right auricle, causes its dilatation; while the auricle so dilated in its turn retards the flow of blood through the jugular veins, which then exhibit distension. The same effect will, of course, be produced by any obstruction to the return of blood to the heart, whether seated in the lungs themselves, or in the mitral orifice, or the valves which close it.

This distension is necessarily accompanied with more or less of a pulsatory movement in the vein, the blood being only able to reach the heart during inspiration. This, however, is not the only pulsatory movement which the veins in this region exhibit when they are in a state of distension. The systole of the right ventricle causes a vibration which passes through the tightly-stretched right auriculo-ventricular valve, and the thrill thus communicated to the blood in the dilated auricle is thence transmitted to the jugular veins. In this case the tricuspid valve and the valves at the mouth of the jugular veins are competent, and there is, therefore, no backward flow of blood into auricle or vein; it is simply the impulse which is transmitted. This we can readily satisfy ourselves of by compressing the right jugular vein high up in the neck, and then if the contents of the lower part of the vessel be pressed out, the vein will not fill again from below, since no valvular incompetency exists.

When, however, the tricuspid valve is incompetent, or when the valves in the jugular vein cease to close the lumen of that vessel (either from destruction of its valves, or from extreme dilatation of the vein preventing the valves from doing their duty), the vein when so emptied will be seen to fill from below with regular pulsations corresponding to those of the right ventricle.

Thus is formed the "venous pulse," one of the most important signs of tricuspid incompetence.

Jugular pulsation may occasionally be præsystolic in rhythm, the movement resulting from the transmission of the impulse of the auricular systole into the vein.

Sudden collapse of the jugular veins during the ventricular diastole has been shown by Friedreich to be a sign of pericardial adhesions.

3. *Epigastric Pulsation* may be conveniently divided into two groups (1) those which are synchronous with the ventricular systole; (2) those which follow that systole after a slight but appreciable delay.

(*a.*) *Synchronous with the Ventricular Systole.*—When the right ventricle is hypertrophied and dilated, it may frequently be felt to pulsate in the epigastrium, and any condition which depresses the diaphragm or forces the heart towards the right may give rise to this pulsation.*

The liver may also pulsate in the epigastrium, but if the impulse is exactly systolic in rhythm, it can only be occasioned by direct transmission from the adjacent right ventricle.

(*b.*) *Delayed Epigastric Pulsation, i.e.*, that which succeeds the ventricular systole after an appreciable interval, may be due to the transmitted impulse of the abdominal aorta. The pulsation is then somewhat to the left of the middle line; it extends downwards towards the umbilicus, and is not diffused laterally. It may be conducted to the parieties by means of tumors, or through the overlying liver. The pulsation may be due to an aneurism on the abdominal aorta, or one of its branches, when it will have a distensile character.

The venous pulsation which has been already noticed as occurring in cases of incompetence of the tricuspid valve is not limited to the jugular veins, it also takes place in the inferior *vena cava*. This pulsation may be communicated to the liver, and if the hepatic veins be likewise affected, pulsation becomes not merely heaving but distensile.†

In all these conditions the pulsation follows the apex-beat after a slight interval of time. The delay can be best appreciated by fixing with wax, over each pulsating point, a bristle carrying a small flag.

Systolic indrawing of the epigastrium occurs rarely, and is caused by extensive pericardial adhesions.

---

* Rosenstein has proposed to call this pulsation "parepigastric," as it lies rather at the border of the left ribs than in the centre of the epigastrium.

† To discriminate the various epigastric pulsations mentioned requires the use of palpation as well as inspection, but to preserve the continuity of the subject, they are all grouped together in this chapter.

TABULAR STATEMENT OF VARIOUS EPIGASTRIC PULSATIONS.

1. Synchronous with the ventricular systole.
   (*a.*) Pulsation of the left ventriclee.
   (*b.*) Pulsation of the liver transmitted from the left ventricle.
2. Delayed.
   (*a.*) Aortic pulsation (hysterical or other.)
   (*b.*) Aortic pulsation transmitted through the liver, overlying tumors, etc.
   (*c.*) Aortic or other aneurisms in epigastrium (distensile).
   (*d.*) Pulsation of the inferior *vena cava* in cases of tricuspid incompetence transmitted through the liver.
   (*e.*) Distensile hepatic pulsation arising from a venous pulse in the hepatic veins in cases of tricuspid incompetence.

4. *Arterial Pulsations on the Thoracic Wall.*—Aortic aneurisms frequently give rise to visible pulsation in the upper part of the thorax, above the third rib. Such pulsation is systolic in rhythm, being as nearly as possible synchronous with the ventricular systole. If the ascending portion of the arch be involved, the pulsation usually lies to the right of the sternum; if the transverse portion be the seat of the disease, the pulsation is more in the middle line; and if the aneurism affect chiefly the descending part of the arch, the pulsation lies to the right side of the sternum. Aneurisms of the innominate and subclavian arteries also give rise to visible pulsation in the walls of the thorax. Systolic pulsation at the second left interspace is sometimes communicated to the surface from the subjacent pulmonary artery in cases of retraction of the borders of the lungs.

A diastolic impulse is sometimes to be seen and felt over the seat of the aortic and pulmonary valves (second right and second left intercostal spaces) in very emaciated persons, especially where the borders of the lungs have become retracted.

5. *Capillary Pulsation* can occasionally be observed on the cheeks, beneath the nails, or in the line of congestion caused by drawing a sharp point, such as that of a pencil, over the skin, and though sometimes occurring independently of that cause, is usually due to incompetence of the aortic valves, with hypertrophy of the left ventricle.

CHAPTER IX.

Circulatory System.

PALPATION.

Cardiac Impulse. — Altered in position — Vertically — Horizontally — Altered in force—Altered in Extent—Thrills, Endocardial and Exocardial.

The skilled application of the hand to the cardiac region gives important information regarding the heart, which inspection alone is not fitted to communicate, and confirms much that inspection has already indicated. Palpation deals chiefly with the phenomena of the cardiac impulse, and with the various thrills which sometimes occur in connection with the heart's action.

CARDIAC IMPULSE.

The apex-beat has already been spoken of in the last chapter in its normal condition. We now consider the various changes which it may undergo in disease—alterations in position, in strength, and in extent—remembering that in health the impulse lies between the fifth and sixth ribs, about two inches to the left of the sternum; that while ordinary respiration does not affect its position, it is depressed and elevated to a very slight extent by deep inspiration and expiration; and that when the patient lies on the left side it is slightly displaced outwards.

Alterations in the position of the Apex-beat.

1. *Vertically.*

The height depends simply on the level of the diaphragm.*

Pulmonary emphysema and spasmodic contraction of the diaphragm cause a general depression of the diaphragm, while collections of liquid or of gas in one pleural cavity, or in the pericardial sac, an increase of the weight of the heart (particularly from hypertrophy of the left ventricle), or the presence of tumors in the neighborhood of that organ, produce a local depression in the diaphragm, and each of

* With the exception of cases of pericardial effusions, when the diaphragm may be depressed and the apex-beat simultaneously raised.

these conditions finds expression in a lowered position of the apex-beat.

On the other hand, if the diaphragm be raised owing to cirrhosis of one lung, or contraction following the absorption of a pleural effusion, or by reason of increased abdominal pressure, the result of tumors, ascites, meteorism, etc., the apex-beat will be correspondingly elevated.

2. *Laterally.*

(*a.*) *To the right.*—Hypertrophy and dilatation of the right ventricle displace the apex-beat from its normal position toward the right. In slighter cases the pulsation is greatest in the epigastrium, but in those which are more marked, the apex, or at any rate that portion of the heart-wall which represents the apex, beats under the right edge of the sternum, or even farther to the right. Effusions in the left pleural cavity also push the heart over to the right side; and on the absorption of the exudation the heart returns again to its normal position, provided that it has not become bound down by adhesions in its abnormal position. The apex-beat may be found on the right side of the thorax in cases of congenital transposition of the viscera.

(*b.*) *To the left.*—Hypertrophy and dilatation of the left ventricle not only depress the apex-beat, but also move it considerably to the left. In cirrhosis of the left lung the heart follows the contracting lung towards the left, and effusions into the right pleural cavity also produce a movement of the apex-beat in this direction, when they are of considerable amount.

Strength of the Apex-beat varies much even in healthy individuals, owing to varieties in the thickness of the chest-wall, in the width of the intercostal spaces, and in the extent to which the apex is overlapped by pulmonary tissue. Pathologically, the differences are still more apparent.

Diminished force of the cardiac impulse, even to the extent of being imperceptible to the finger, may be due to—

1. *Intrinsic causes.*—These include abnormal innervation, fatty degeneration of muscular fibre of the heart, and that inflammatory change which follows myocarditis and hyperpyrexia of long duration, and lastly, deficiency of proper blood supply and interference with the circulation.

2. *Extrinsic causes.*—When the visceral and parietal layers of the pericardium become adherent, the movements of the apex of the heart are so interfered with that all cardiac

impulse may be lost. Effusions of fluid or gas into the pericardium or into the left pleural cavity, as well as intervention of emphysematous lung tissue between the heart and the thoracic wall, all tend to weaken the apex-beat.

Increase in force of the cardiac impulse may be of neurotic origin; it may be the result of fever, of endocarditis, of pericarditis, or of any condition which is more than usually favorable to the conduction of the impulse, but by far its most common cause is hypertrophy of the heart. The heaving impulse which results from hypertrophy of the left ventricle is much more easily detected than that which hypertrophy of the right ventricle occasions, as the latter has its point of maximum intensity behind the sternum.

The *rhythm* of the cardiac pulsations will be noticed in the remarks made on the arterial pulse.

The Extent of the Impulse.—Normally, the apex-beat is not perceptible over more than an area of about an inch in breadth, and is limited to the fifth intercostal space. When the pulsation extends much beyond such limits, it is abnormal. In disease the apex-beat not unfrequently becomes diffused over a considerable area, and this may result from increased action of a normal heart (medication, excitement, etc.); from cardiac hypertrophy; from the application of an unusually extensive area of the heart to the thoracic walls (retraction of the lungs); or merely from great thinness of the chest-wall.

Double Apex-beat.—Independent, non-synchronous contraction of the ventricles has been met with in a few rare cases (Charcelay, Leyden, Roy, etc.), where the systole of the right ventricle caused a pulsation in the jugular veins which alternated with that in the carotid arteries.

Thrills may be felt by the hand applied over the cardiac region, and these are of two kinds—

1. *Endocardial thrills*, caused by the blood current being forced through a small opening. The pathological condition which gives rise to the thrill is indicated by the seat of greatest intensity and the time in relation to the various phenomena of cardiac action.

Thrills in the mitral area (a circle with a radius of one inch around the apex-beat) are systolic or præsystolic according as they are produced by incompetence of the mitral

valve or stenosis of the orifice which that valve covers. Thrills over the second right costal cartilage arise from aortic stenosis or incompetence; in the former case being systolic, and in the latter diastolic in rhythm. Præsystolic and systolic thrills in the tricuspid area indicate respectively stenosis of the orifice and incompetence of the valve. Very rarely a systolic thrill is felt over the pulmonary artery, denoting stenosis of the pulmonary orifice, or a diastolic, indicative of regurgitation.

2. *Pericardial friction-fremitus*, caused by the rubbing during the heart's action of the two pleural surfaces which have been rendered rough and uneven by the effusion of lymph; more readily detected by auscultation than by palpation.

CHAPTER X.

CIRCULATORY SYSTEM—(*continued*).

PERCUSSION.

THE heart, lying in the thoracic cavity, has, in its normal condition, the following relations to the anterior wall of the chest:

The *right border*, formed almost entirely by the right auricle, stretches in a slightly curved manner from the second right intercostal space downward and outward to the cartilage of the fifth rib on the same side, just at its junction with the sternum. The *left border*, formed by the left ventricle, reaches from the second intercostal space on the left side downward and outward to a point about half an inch outside of the apex-beat. The *lower border* corresponds pretty exactly to a line joining the sternal end of the fifth right costal cartilage with the apex-beat. The highest portion of the heart, formed by the appendix of the left auricle, reaches a level which may be indicated by a line joining the lower border of the sternal ends of the second pair of costal cartilages.

To percuss the heart it is best to have the patient recumbent. No strong percussion is, as a rule, required, and this is specially the case with regard to the absolute dulness

where the tap should be light. In addition to the note elicited, the sense of resistance perceived during percussion is of great value.

The greater portion of the heart is separated from the chest-wall by the overlapping lung, and therefore it is only the small uncovered part, consisting of the right ventricle, which yields on percussion a dull note. This area of *absolute cardiac dulness* is readily mapped out. Its right border extends along the right margin of the sternum (or along the mid-sternal line in some cases) from the level of the fourth to that of the sixth costal cartilage. The left border is an irregular line stretching from the upper end of the right border to the apex-beat. The lower edge cannot be defined by percussion, because at this point the cardiac merges into the hepatic dulness. Its position can, however, be obtained with approximate accuracy by drawing a line from the upper limit of absolute hepatic dulness on the right side to the apex-beat. The area thus formed is roughly quadrilateral in shape ; and not only do its extent and position vary with each respiration, but they are also affected by the position of the person, and also by the constant changes of the diaphragm in respiration, necessarily producing corresponding changes in the lie of the heart. The condition of the margins of the lungs (as in emphysema) have also an important influence on the size and position of the area of absolute cardiac dulness. The variations to which this area is thus liable greatly diminish the value of its condition as indicative of disease.

The region of absolute dulness is *increased* in—

(1.) *Hypertrophy and Dilatation of the Left Ventricle*, when the increase takes place chiefly downward and to the left.

(2.) *Hypertrophy and Dilatation of the Right Ventricle.*—In this case the left margin is little interfered with, while the right is thrown outward toward the right.

(3.) *Serous Effusion into the Pericardium.*—The dulness here takes a pyramidal form, being limited by the pericardial sac, with the base downwards, resting on the diaphragm.

(4.) Increase of absolute cardiac dulness may be simulated by various pathological conditions of the neighboring organs, such as infiltration of the margins of the lungs, pleuritic effusions, etc.

It must be borne in mind, however, that these various diseased conditions of the heart and its investing sac may

be present without giving rise to any appreciable changes in the absolute cardiac dulness. The area of absolute dulness is diminished, or entirely lost in—

(1.) *Left pneumo-thorax*, where the collection of gas in the left pleural cavity is so great as to force the heart to the right. The area of dulness is usually, in such cases, still to be detected to the right of the sternum; but it is much diminished in size.

(2.) *Emphysema*, when well pronounced, entirely does away with absolute cardiac dulness, the margins of the lungs approaching so near to one another as to overlap the heart completely. In slighter cases the area is only diminished in size.

(3.) In the rare cases in which free gas is found in pericardium (pneumo-pericardium) percussion elicits a clear note over the whole cardiac area.

If, then, as has been said, the indications regarding the heart itself obtained by examination of the area of absolute cardiac dulness are vitiated by various pulmonary conditions, we must seek to obtain information from the percussion of that portion of the heart which is covered by a more or less thick layer of lung.

In percussing the thorax, as we pass toward the cardiac area, the note, which is at first purely pulmonary in character, grows more and more dull, until the limit of absolute cardiac dulness is reached. The reason of this change in the note will be fully discussed when the subject of percussion comes to be treated of as a whole in a subsequent chapter.

It is by noting the point at which this change in the note occurs that the position of the outer margin of the heart can be determined by percussion. This is called the area of *relative cardiac dulness*. Now, although it is in most cases not difficult to sketch out the entire area of relative dulness, yet for ordinary clinical purposes it is only necessary to percuss in two directions—vertically, parallel to the left margin of the sternum, and transversely, at the level of the fourth rib.

1. *The vertical line* is thrown about one inch to the left of the sternum in order to avoid the aorta. In this direction the full note of the lung becomes impaired at about the lower edge of the third rib. This is, then, the limit of relative cardiac dulness in this situation. Prolongation of dulness upwards above the third rib (if it be not caused by dislocation of the heart upward, by pulmonary consolida-

tion, or by the bulging of an aortic aneurism) arises from pericardial effusion. On the other hand, increase of dulness at the *lower* end of this parasternal line (if the heart be not dislocated downward) indicates enlargement of the left lobe of the liver.

(2.) *The transverse line* at the level of the fourth rib. The *left border* of the heart is usually marked with sufficient accuracy by the position of the apex-beat, but in cases of uncertainty it may be determined by percussing from without inward in the direction indicated, when it will be found that the margin of relative dulness lies a short distance ($\frac{1}{2}$ to 1 inch) to the left of the absolute cardiac dulness. Increase of the dulness to the left usually indicates hypertrophy and dilatation of the left ventricle. The *right margin* of relative dulness at the level of the fourth rib indicates the amount of dilatation of the right auricle. It is usually found about one inch to the right of the margin of the sternum. Increase of dulness in this direction therefore takes place when the right auricle is distended, and indicates obstruction to the circulation. This obstruction is probably aortic if the apex-beat is displaced downward and outward. If this be not the case, it is either mitral or pulmonary.

The aortic dulness exists in the normal condition simply as a slight rounded projection from the upper part of the relative cardiac dulness. It rises as high as the upper margins of the second costal cartilages.

When dilatation of the aortic arch takes place, the relative dulness becomes better marked, and passes upwards and towards the right, impairing the note over the *manubrium sterni*. In aneurism of the aortic arch, the area of dulness increases correspondingly, and if the tumor approach near to the sternum there is produced an area of absolute dulness.

When the aneurism comes to press firmly on the breastbone, the dulness which this gives rise to is not absolutely limited to the site of the tumor, but extends up and down the sternum to a variable distance. This dulness is probably in some measure caused by the obstruction to the vibration of the sternum which the firm pressure of the tumor presents. A similar alteration in the percussion note may be artificially produced by pressing heavily on the sternum with the hand while percussion is being made at a neighboring point.

CHAPTER XI.

CIRCULATORY SYSTEM—(*continued*).

AUSCULTATION OF THE HEART.

CARDIAC auscultation is almost invariably practised with the aid of a stethoscope. For ordinary purposes a simple wooden instrument suffices, the cup of which accurately suits the ear of the auscultator; but in exceptional cases a double stethoscope, such as that of Alison, may be used, which fits into both ears.

On listening over the heart two sounds are to be heard, separated by two pauses of unequal length. The first sound, which is considerably the more prolonged of the two, is followed by a short pause: to it succeeds the short second sound, and finally a long pause. At the apex of the heart the first sound is the louder of the two, the rhythm being there trochaic (¯˘), while at the base the accent is thrown upon the second sound, as in the iambus (˘¯).

Associated more or less intimately as these sounds are with the valves of the heart, it is necessary for the student to have a clear conception of the position which these structures occupy in relation to the anterior thoracic wall.

The Mitral Valve, which is situated on a plane considerably posterior to those in which the other valves lie, may be said to correspond to the sternal end of the third left costal cartilage, projecting more or less upward and downward into the adjacent intercostal spaces.

The Tricuspid Valve.—The attached edge of this valve corresponds to a line drawn slantingly across the sternum from the third left intercostal space to the fifth right costal cartilage.

The Aortic Valves lie horizontally, opposite a line joining the middle of the sternum and the inner end of the third left costal cartilage.

The Pulmonary Valves are also placed horizontally, slightly higher and more to the left than the aortic, corresponding to the upper border of the third left costal cartilage, or to the second left interspace.

It will thus be seen that, in relation to the chest-wall, these valves lie very close to one another, a superficial area

of half an inch square including a portion of all four (Walshe). The sounds produced in connection with these valves are, however, best heard, not immediately over them, but at that point on the chest-wall at which the cavity into which the vibrating blood is flowing approaches nearest to the surface. Naturally, this point where the sound is most intense varies in each case, and hence we have four areas for auscultation.

The *Mitral Area* is a circle about an inch in diameter, surrounding the apex-beat. This is the only point at which the left ventricle comes in contact with the chest-wall.

The *Tricuspid Area* embraces the lower part of the sternum, particularly the left border at the level of the fourth, fifth, and sixth cartilages.

The Aortic Area.—The aorta approaches nearest to the chest-wall at the second right costal cartilage, and consequently the aortic area is situated at this point.

The Pulmonary Area corresponds to the second left intercostal space (Von Dusch), or to the third left costal cartilage.

The cause of the heart-sounds heard in these various areas may be briefly indicated as follows:

The *first sound* (systolic), heard in the mitral and tricuspid areas and synchronous with the ventricular systole, is formed partly in connection with the left ventricle, and partly in connection with the right. In each case its mode of production is similar, and depends (in all probability) upon two factors. The first of these, as was shown by Rouanet as early as 1832, is the vibration of the auriculo-ventricular valves, arising from the sudden tension caused by the ventricular contraction; and the second is the muscular vibration in the muscular fibre of the ventricle, which takes place during its contraction (muscle sound).

The first sound, as heard in the aortic and pulmonary areas, is probably to be considered as due to the conduction of the mitral and tricuspid sounds to the base of the heart.*

The *second sound* (diastolic) heard in the aortic and pulmonary areas has its origin in the vibrations produced in the semilunar valves at the orifices of each of these vessels

---

*According to Heynsius, however, the first sound, at the base of the heart, arises independently at the orifices of the aorta and pulmonary artery, and is the result of vibrations arising in the blood currents which are propelled into these two vessels during the ventricular systole.

during the diastole of the heart, by the blood being forced back upon these valves.

The second sound, as heard in the mitral and tricuspid areas, is merely the aortic and pulmonary sound conducted to the apex.

It is evident that we have thus to deal with four sounds during the course of the cardiac revolution, which arise entirely independently of one another.

Two systolic sounds originate at the mitral and tricuspid valves, and in the muscular fibre of the ventricles; and two diastolic sounds are caused by vibration of the semilunar valves at the aortic and pulmonary orifices.

Of these, the first two are synchronous, and are consequently heard as one sound, and, as the last two also take place almost simultaneously, only two sounds, a systolic and a diastolic, are audible over the cardiac area. The systolic sound marks accurately the commencement of the ventricular systole, and the diastolic expresses with equal precision the instant at which the diastole of the heart begins.

The changes in the cardiac sounds which disease produces are of two varieties—(1) Alterations in the normal heart sounds; and (2) murmurs, or adventitious and abnormal sounds.

MODIFICATIONS OF THE NORMAL HEART SOUNDS.

The normal sounds may be modified in disease as regards intensity, purity, quality, etc. It will suffice to consider such changes under the three following heads:

1. **Variations in Intensity** (intensified or enfeebled)—

(*a.*) *Intensified.*

This takes place with regard to all the heart sounds under the influence of mental or bodily excitement, pyrexia, etc., or in emaciated persons, simply on account of the thinness of the chest-walls, or finally, as a result of improved conduction occasioned by condensation of the pulmonary tissue, or by the bulging of an aortic aneurism.

When the accentuation is limited to one sound, as heard in a particular area, it may result (1) from the better conduction through consolidated lung-tissue (particularly in the case of the second sound at the base of the heart), (2) from hypertrophy of the walls of a particular cavity of the

heart, or (3) it may arise from increased tension in the column of blood which presses back on the valves.

Intensification is most significant when found in connection with the second sound in the aortic or pulmonary areas. In order to determine that either of these sounds is accentuated it is only necessary to compare it with the other, always bearing in mind, however, that in the normal condition the aortic sound is louder than the pulmonary.

Accentuation of the aortic second sound arises from increase of the arterial tension. It is also met with in dilatation of the aortic arch, and is most marked in cases of aneurism of the arch, where there is hypertrophy of the left ventricle, but in which the sigmoid valves are still competent.

Accentuation of the pulmonary second sound is found wherever there is increased pressure in the pulmonary artery. It is a sign that some hindrance exists to the circulation in the lungs, and it is well marked in cases of mitral stenosis and mitral incompetence.

(*b.*) *The sounds may be enfeebled* by reason of bad conduction through thick chest-walls, emphysematous lungs, pleural effusions, etc., or they may be audible with difficulty on account of loud sounds in the neighboring lungs. Weakness of particular sounds is chiefly seen in connection with affections of the myocardium, as after typhus, in fatty degeneration of the cardiac muscle, etc.

2. **Impurity of the Sounds.**—A heart sound is said to be impure when it wants the clearness and definition of normal sounds, or when it consists of or is accompanied by irregular vibrations. Such slight changes do not amount to a murmur, but in practice an impure sound is not readily distinguished from one accompanied by a soft murmur.

Impurity in the heart sounds may be caused by thickening of the valves, irregular tension, and closure of the different cusps of the valve.

Impurity is of little diagnostic importance.

3. **Reduplication of the Heart Sounds.**—Not uncommonly the heart sounds become doubled, each cardiac cycle giving rise to three, or even four, separate sounds. On careful examination, it will be found that one or other sound has become broken up into two. Reduplication frequently occurs in health, and is then intimately associated with respiration, and the changes in the intrathoracic pressure thereby

produced. The first sound is reduplicated at the end of expiration and the beginning of inspiration, while the second sound is doubled at the end of inspiration and the beginning of expiration. Such reduplication has no peculiar significance, and only indicates that, in the case of the first sound, the auricular pressure on the right side of the heart is increased, and retards the closure of the tricuspid valve; and, in the case of the second sound, that the pressure in the pulmonary artery is increased, and so the closure of the pulmonary valves is accelerated.

In disease, reduplication is more marked and constant. It is frequently found in connection with the second sound in cases of mitral constriction and lung disease, from the abnormally high tension in the pulmonary artery thereby produced. It may also arise in cases of aortic stenosis. Reduplication of the first sound may be produced by irregular contraction of capillary muscles, or, in very rare cases, by a synchronous contraction of the ventricles, in addition to its origination from differences of blood tension as in health.

MURMURS.

The murmurs which are met with in connection with the heart's action are divided into two groups: (1) *Endocardial*, or those which arise within the heart; and (2) *Exocardial*, or those originating in connection with the outer surface of that organ.

1. Endocardial Murmurs.—Murmurs, or abnormal sounds, differ from the natural heart sounds by being more prolonged, and less sharply defined. Those which are of endocardial origin all arise from oscillations or vibrations in the blood stream, as it passes through a narrow opening into a wider space beyond.

To go more particularly into the physical question of the origin of murmurs, we must put away from our minds the idea that such murmurs are ever caused by rubbing of the blood stream upon roughnesses or irregularities on the valves or orifices of the heart. Such a state of matters is physically impossible; for when a fluid streams through a tube, *the walls of which it wets* (as blood does the endocardium), a thin layer of the fluid becomes attached to the inner wall of the tube by the force of cohesion, and consequently, seeing that the current itself never comes in con-

tact with the tube-wall, no friction between the two is possible (Neumann, Helmholz, etc.)

When a fluid is passing along a tube of uniform calibre no murmur arises, unless the rapidity of flow is very great. The blood current is never rapid enough to give rise to a murmur under these conditions. But when a constriction exists in the tube, and the fluid is thus forced to pass from a narrow into a wider portion, a murmur readily arises; and the greater the difference between the lumen at the two points the more easily is the murmur produced; or, in other words, the less rapidity of current is required for its production.* It is in this way, and under such conditions, that all such cardial murmurs arise. Whenever the blood stream passes with sufficient velocity through a narrow orifice into a wider space beyond, there will be such friction between the fluid particles as to give rise to sonorous vibrations in the fluid, as when, for example, a rent occurs in the aortic or mitral valves, or when the orifices they guard become narrowed by disease.

In the production of murmurs there enters, however, another factor which must not be entirely lost sight of, viz. —the condition of the blood. So early as 1837 it was shown† that the ease with which murmurs could be produced by driving fluids of different density through veins stood in inverse ratio to the density of the fluid, and this observation has been confirmed by many subsequent experimenters, among whom may be mentioned Weber,‡ Nolet,§ and Richardson.‖ It was found, in particular, that when water was added to blood a murmur was more easily produced than when blood alone was used, and this observation has a very important bearing upon anæmic murmurs, and particularly those which arise in veins, as will be hereafter shown.

Endocardial Murmurs are of two varieties—(1) Those of valvular origin, and (2) those of other than valvular origin.

Of these, the latter class is so rare that it is well invariably to endeavor to associate a murmur with a particular

---

* That such murmurs are not due to the vibration of Savarts's "veine fluide" is shown by the fact that the murmur may arise in tubes of uniform calibre where no such "veins" are formed.

† *Vide* Williams, on the *Pathology of Diseases of the Chest.* London, 1840.

‡ *Arch. f. physiol. Heilkunde*, xiv.

§ *Arch. der Heilkunde*, xii. 1871. ‖ *Med Times and Gazette*, 1868.

valve or orifice, and only in the event of failure in such an attempt, to consider the possibility of a non-valvular cause.

Having ascertained the presence of a murmur, there are three points which should be carefully noted—(1) The rhythm of the murmur, *i.e.*, the particular period in the cardiac contraction with which it corresponds (auricular or ventricular systole or diastole, etc.); (2) The point of maximum intensity and the direction of propagation; (3) The condition of the normal heart sound at the valve or orifice at which the murmur is supposed to originate.

(1.) *Rhythm.*—To ascertain the rhythm of a murmur it is necessary to lay a finger upon the apex-beat or upon the carotid artery while we auscultate. This gives the time of the ventricular systole, and enables us to say which is the first and which the second sound, and consequently the rhythm of the murmur can be readily ascertained. If, however, the cardiac pulsations exceed 90 per minute, it may be impossible thus to time the murmur, and in such cases we must wait till rest and appropriate medication have reduced the rapidity of the heart's action.

(2.) *Point of Maximum Intensity and Direction of Propagation.*—It has been already stated that the normal heart-sounds are heard with most distinctness in various areas according to the valve or orifice at which they arise. These sounds are conducted in the direction of the blood current, and are best heard where the cavity into which the current flows approaches nearest to the surface of the body. The same holds good for murmurs, every endocardial murmur of valvular origin having its point of maximum distinctness in one of these four areas, and being of mitral, tricuspid, aortic, or pulmonary origin, according as it is best heard in the mitral, tricuspid, aortic, or pulmonary area. (Two exceptions to this rule however exist, viz.—(1) a mitral systolic murmur, which is sometimes best heard an inch to the left of the pulmonary area, and (2) an aortic diastolic murmur which is occasionally most intense at the xiphoid cartilage.)

Having satisfied ourselves as to the rhythm, and the point of greatest intensity (and consequently the seat of origin) of the murmur, it is a matter of simple reasoning to discover its mode of production. Thus, for example, a systolic mitral murmur can only be one of regurgitation through incompetence of the valve. A præsystolic mitral murmur, on the other hand, must result from stenosis of the mitral

orifice, since it occurs at the instant when the blood is being propelled by the auricular systole through the mitral orifice into the ventricle. We shall consider the causation of each particular murmur farther on.

The direction of the conduction of murmurs may be of use in indicating their origin. Stated generally, it may be said that systolic mitral murmurs are conducted toward the left axilla, and to the angle of the left scapula; tricuspid murmurs are heard over an area corresponding to the right ventricle; aortic murmurs are propagated up and down the sternum and into the arteries; and finally, pulmonary murmurs are usually not audible outside of that area.

(3.) *The Condition of the Normal Sound at the Orifice at which the Murmur originates.*—The presence of a normal sound, more or less obscured by the accompanying murmur, indicates that the valve is not entirely destroyed. The method of auscultation suggested by Gendrin is of value for the purpose of ascertaining this. He recommends the ear to be slightly raised from the stethoscope, the instrument remaining unmoved, when the sound will become more and the murmur less audible. The real value of the presence of a cardiac sound as an indication of the state of the valve is very questionable. In the case of aortic disease, the auscultation of the arteries gives much more reliable results.

The character of the murmur (soft, blowing, rasping, whistling, etc.) is of little diagnostic value. As a general rule, *direct* murmurs (those which arise in the blood current as it is flowing in its normal direction) are rough; whereas *indirect* murmurs (those which flow in a direction contrary to that of the normal current) are soft.

Having determined the rhythm and seat of the murmur, it is, as I have already said, no very difficult matter to infer the manner of its causation. This is done by simply bringing to mind what is happening at the valve in question during the particular period at which the murmur is heard. In order to make this plain we will now consider very briefly the various murmurs met with in connection with each valve.

Mitral murmurs are systolic, diastolic, or præsystolic in rhythm, according as they occur during the ventricular systole, the diastole, or immediately before the ventricular systole (*i.e.*, during the auricular systole.)

Mitral systolic murmurs.—These murmurs, originating at

the mitral valve during the ventricular systole, indicate that from some cause the valve does not completely cover the orifice, but allows a part of the blood contained in the ventricle to be forced back into the auricle. This incompetence of the mitral valve is the result of one of two processes; either the valve itself has become altered in shape, or the orifice has increased in size, so that the valve which formerly sufficed to occlude it is not now sufficiently large.

The former condition arises as a result of subacute and chronic endocarditis (caused in connection with rheumatism, syphilis, child-birth, scarlatina, etc.) The valves become thickened and shrivelled up, and so the incompetence originates.

The second condition (viz., increase in the size of the mitral orifice) is found in all diseases in which relaxation of the cardiac muscle takes place to a marked extent, as in typhus, typhoid, relapsing, and rheumatic fevers; in scarlatina, measles, erysipelas, smallpox; and not least importantly, in chorea and in various forms of anæmia, particularly in chlorosis. In all these conditions, the myocardium is so softened and stretched as to allow the mitral orifice to become too large to be covered by the valve. Regurgitation of blood takes place into the auricle; and in passing from a narrow orifice into a cavity sonorous vibrations arise in the fluid, in the manner already described. In these cases the formation of a murmur is no doubt aided by the watery condition of the blood, in the manner already described.

Mitral systolic murmurs are usually heard with greatest distinctness in the mitral area, and are propagated toward the left axilla and the angle of the left scapula. Occasionally, however, they are most audible a little to the outside of the pulmonary area, especially in the case of anæmic murmurs; and this probably arises (as Naunyn has pointed out) because the sonorous vibrations in the auricle are conducted into the auricular appendix, and become most audible at that point on the chest-wall where the appendix approaches nearest to the surface—*i.e.*, about an inch and a half to the left of the pulmonary area.

Mitral præsystolic and diastolic murmurs arise from the same cause, viz.—stenosis (narrowing) of the mitral orifice. Immediately after the ventricles of the heart have contracted they relax and begin to refill with blood, and during the period of time represented by the second or diastolic

sound, and by the long pause, this process of filling goes on. At first the blood follows the retreating walls of the ventricles, propelled partly by gravity and partly by the ordinary intra-thoracic pressure, and so flows slowly through the patent orifices (mitral and tricuspid) into the respective ventricular cavities. But toward the end of the long pause the auricular connection takes place, and the remainder of the blood is thus more powerfully forced into the ventricles. In ordinary circumstances these actions take place noiselessly; but when stenosis of the mitral orifice arises (we speak now of the left side of the heart alone) as a result of endocarditis, the narrowing may be sufficient to throw the fluid into sonorous vibrations. It depends on the rapidity of flow, and the narrowness of the orifice in relation to the size of the ventricular cavity, whether or not a murmur will occur—if so whether it will be diastolic or præsystolic in rhythm, or in other words, whether it will be produced when the blood is flowing into the ventricle immediately after the ventricular systole, or later on, during the auricular systole.

These murmurs sometimes coexist, and may either run into one another, and so fill up the whole time occupied by the ventricular diastole, or they may be separated by a very short interval of silence. The diastolic portion is usually soft, whilst præsystolic (or auricular-systolic) murmurs are almost invariably rough in character.

Tricuspid murmurs resemble those at the mitral valve in regard to their causation.

Systolic tricuspid murmurs are indicative of incompetence of the valve, with consequent regurgitation of blood into the right auricle during the ventricular systole. This results either from deformity of the valve, produced, as in the case of the mitral valve, by endocarditis, or from dilation of the orifice. The latter condition may be occasioned by such causes as produce a corresponding state of matters on the left side of the heart (fevers, anæmia, etc.), but more commonly this relative incompetence, as it has been called, is caused by distension of the right auricle and ventricle, the result of obstruction to the circulation through the lungs, produced most markedly in stenosis, or incompetence of the mitral valve.

Præsystolic tricuspid murmurs are very rarely met with, and never without other valvular complications. They are the result of stenosis of the tricuspid orifice, and the mechanism

of their production is similar to that which produces the corresponding mitral murmur.

Aortic murmurs are of two varieties—systolic and diastolic. These usually coexist.

Systolic aortic murmurs are those produced at the aortic orifice as the blood is propelled into the aorta by the contraction of the left ventricle. Such a murmur arises when the orifice is contracted or roughened as a result of endocarditis. The murmur is usually loud and sawing, occasionally musical, and whilst it is loudest in the aortic area, it can most frequently be heard over the whole front of the heart.

Diastolic aortic murmurs are the result of incompetence of the aortic valves, the blood regurgitating from the aorta into the left ventricle during the ventricular diastole. The position of maximum intensity of this murmur varies very much. In many cases it is best heard in the aortic area; not uncommonly it is loudest at the ensiform cartilage; rarely the apex-beat is the situation at which it is most distinct.

Most usually these two murmurs are heard together, the so-called double aortic murmur, for the valves are rarely incompetent without presenting some obstruction to the flow of blood over them into the aorta.

Pulmonary murmurs.—Among these we do not include those hæmic murmurs which arise at the mitral valve, and have their seat of greatest intensity an inch or more to the left of the pulmonary area.

True pulmonary murmurs are of very rare occurrence. They are systolic and diastolic in rhythm.

Systolic pulmonary murmurs are either inorganic or organic. The former have been supposed by Quincke to be produced where from some cause the left lung is retracted, and the heart in its systole so compresses the pulmonary artery as to give rise to sonorous waves in that vessel.

Organic systolic murmurs are almost invariably due to congenital constriction of the pulmonary artery. Such cases are rare, and differ much from one another according to the period of cardiac development at which the constriction commenced. The ventricular septum is usually deficient, with cyanosis as a consequence.

Diastolic pulmonary murmurs are still more rare. They result from incompetence of the pulmonary valves, and are invariably accompanied by systolic pulmonary murmurs.

2. *Endocardial murmurs of non-valvular origin* are probably of very rare occurrence indeed. They may result from—

(1.) Congenital deficiency of some part of the septum, which divides the two sides of the heart; and in that case they only intensify the valvular murmurs already existing.

(2.) Flakes of lymph attached to the valves are said to cause such murmurs.

(3.) Changes in the density of the blood in anæmia, chlorosis, etc., may allow of murmurs forming under conditions under which no such sonorous vibrations would arise in blood of normal composition. It has been already pointed out that many of the hæmic murmurs are mitral in their origin, resulting from incompetence caused by relaxation of the cardiac muscle. A small proportion of these murmurs may, however, arise in the blood-stream, where no incompetence exists. Such murmurs are soft, invariably systolic, and usually heard most distinctly over the base of the heart.

2. **Exocardial Murmurs.**—These murmurs are caused by the friction of the two pericardial surfaces on one another, when these surfaces have become roughened as a result of pericarditis, etc. Such friction murmurs are, for the most part, readily distinguished from endocardial murmurs. They are rough and grating, never blowing. They are localized, and are not propagated in the direction of the blood current; and as they usually arise first toward the middle of the heart, the point of greatest intensity does not generally coincide with any one of the cardiac areas. They can always be perceived by the hand, if at all intense, which is only exceptionally the case as regards endocardial murmurs. Further, the rhythm of exocardial murmurs is irregular. They are not confined to any particular phase of the cardiac action, are neither permanently systolic nor diastolic, but vary from minute to minute.

Exocardial murmurs are also sometimes occasioned by friction of two roughened surfaces of the pleura overlying the heart on one another. Such friction murmurs vary in intensity with the movements of respiration.

CHAPTER XII.

CIRCULATORY SYSTEM—(*continued*).

THE EXAMINATION OF THE ARTERIES CAPILLARIES, AND VEINS.

ARTERIES.

THE physical examination of the arteries may be conducted by means of inspection, palpation, percussion and auscultation. Of these we will speak in their turn.

Inspection.—In health the pulsation of the arteries of the body is but little visible, except under the influence of mental emotion or bodily strain. As the result of disease, however, pulsation may become visible in all the superficial arteries of the body, particularly in the carotid, temporal, and radial vessels. All disturbances of cardiac innervation, such as arise in Graves's Disease, and all feverish conditions, are liable to produce such excited action of the heart as will occasion this visible pulsation. Still more marked is the pulsation when the left ventricle is hypertrophied, and, above all, when the aortic valves have been rendered incompetent. Dilated, tortuous, and visibly pulsating temporal or radial arteries are usually found to have undergone atheromatous changes ; and finally, inspection may show us the localized pulsation of aneurism.

Palpation of the arterial system is almost confined to the radial artery, the carotid, brachial, and femoral being but rarely palpated.

The radial pulse is, in health, equal on the two sides; but abnormal distribution, compression, or other pathological condition may so act as to make one pulse weaker than the other. So, also, the pulse-wave propagated from the heart outwards toward the periphery may not arrive at the two wrists synchronously. This condition occurs where there is simple or aneurismal dilatation of the aortic arch, and is particularly noticeable if the aneurism be situate between the innominate and the left subclavian. We may further notice that in this affection the interval of time which oc-

curs between the cardiac systole and the arrival of the blood-wave at the wrist is considerably longer than usual. Such delay arises either from stenosis of the aortic orifice rendering the systole slow and difficult, or from aortic incompetence where (as Tripier has pointed out with great probability) the onward wave meets with, and is delayed by, the regurgitating blood.

It will probably conduce to greatest clearness if the conditions of the pulse are considered under three headings—viz., (1) frequency, (2) rhythm, (3) character.

1. *Frequency of the pulse*, which in the male adult averages about seventy beats per minute (slightly higher in women), varies in healthy individuals according to the age, according to the time of day, the external temperature, and may be greatly influenced by mental emotions and by the administration of certain drugs. In disease the pulse is sometimes abnormally slow, as for example, in jaundice, in fatty degeneration of the heart, and in some affections of the brain. More frequently, however, the pulse rate is increased in rapidity. The rapid pulse of fever, of collapse, and of the various cardiac neuroses is well known. Very generally the pulse is rapid in diseases of the valves of the heart (particularly the mitral).

2. *Rhythm.*—The radial pulsations, which are normally separated by regular intervals of time, and so are rhythmical, may be altered in this relation to each other in a great variety of ways, the normal rhythm being sometimes changed into total irregularity; while at other times the beats, although following each other in an abnormal manner, still possess a certain rhythm. Amongst the latter may be mentioned the (1) *pulsus bigeminus*, in which each two beats form a group separated from the two which precede and the two which succeed by longer pauses than the interval which separates each pair. (2) The *pulsus paradoxus* is that variety of pulse, so carefully described by Kussmaul, where with each inspiration the pulse-wave becomes smaller, or is completely lost. When it is present in all the arteries of the body, it may be due to one of two causes—either to fibrous adhesions between the aorta and the sternum, or some other obstruction which, during inspiration, prevents the free passage of the blood into the aorta; or it may result from any obstruction to the entrance of air into the lungs, which during inspiration lessens the pressure within the thorax. When the *pulsus paradoxus*

occurs only in one radial artery, it is due, as Weil has pointed out, to inflammatory adhesion between the pleura and the subclavian artery. In the *pulsus alternans* there is a regular alternation between a small and a large pulsation. When, after a series of regular pulsations, one or more beats are omitted, the pulse is said to be *intermittent.* These intermissions, due either to momentary cessation of the heart's action or to the blood-wave in question being too feeble to reach the wrist, may be regular or irregular, and often occur independently of heart disease. Most frequently, however, the intermittent pulse is associated with some cardiac affection, generally mitral disease. Very irregular pulsations, in which no rhythm of any kind can be detected, are commonly (although by no means always) due to affections of the mitral valve, generally to mitral constriction, of which affection an extremely irregular pulse, even in the early stages, is an important symptom, and one to which considerable diagnostic importance may attach.

3. *The character of the pulse* varies in a great number of ways, giving rise, especially in the works of the older writers, to a very extensive nomenclature. It will be sufficient for ordinary purposes to notice the following points:

(*a.*) The *expansion* of the pulse. A pulse which reaches its full expansion quickly, and as rapidly collapses again, giving to the finger the impression of a very quick stroke, is denominated the *pulsus celer*, and this celerity is, as Corrigan first pointed out, most distinct where there is aortic incompetence (hence called Corrigan's pulse). The opposite condition, the *pulsus tardus*, is distinguished by the slow manner in which the artery fills and empties, and this sluggishness may be due to slowness in the contractions of the heart, to a hindrance in the capillary and venous circulation, or to loss of elasticity in the arterial wall itself. It is perhaps most frequently met with as a result of arterial sclerosis.

(*b.*) The *tension* of the pulse, or, in other words, the blood-pressure on the inner surface of the artery, may be approximately estimated by the pressure of the finger required to obliterate the pulse. When the tension is high (as in hypertrophy of the left ventricle, lead colic, peritonitis, etc.), we speak of a *hard* or *tense* pulse, and under the reverse circumstances (as in mitral disease), of a soft and compressible pulse. Above all things, however, it must be borne in mind that the impression of tension or hardness may be

given to the finger by a rigid condition of the arterial wall, and it is only when this factor can be eliminated that any safe deductions can be drawn regarding the blood-pressure itself. When the radial artery has undergone calcification, the irregular prominences can usually be felt, and this will prevent error.*

(*c.*) The *volume* of the pulse. A full pulse may be produced by one or more of three factors: powerful ventricular contraction, loss of elasticity of the arterial well, and interference with the blood flow from the arteries into the capillaries. The opposite conditions may give rise to an empty pulse. The pulse is also spoken of as large or small, tremulous, thready, etc.

All these varieties of pulse are best studied with aid of the sphygmograph.

Percussion of the Arteries is almost entirely limited to cases of thoracic aneurism, of which mention has been already made.

Auscultation of the Arteries.

1. *In Health.*—As in cardiac auscultation, so also in auscultation of the arteries, we have to distinguish two phenomena—sounds and murmurs. In health, if the stethoscope be placed over the carotid artery as lightly as possible, two sounds are usually to be heard, corresponding respectively to the expansion and contraction of the artery. Of these the latter is simply the second aortic sound conducted into the carotid, and it seems most probable (Weil, Heynsius†) that the sound coinciding with the arterial expansion ought also to be regarded as the conducted aortic systolic sound (Guttmann,‡ however, regards it as in part originating in vibrations of the arterial wall). These two sounds can also generally be heard in the subclavian; and occasionally the first can also be detected in the abdominal aorta, the brachial, and the femoral; but in the more peripheral vessels no auscultatory phenomenon is present in health. If pressure be made with the stethoscope upon an artery, such as the brachial just above the elbow, where normally no sound can be heard, the narrowing of the lumen of the vessel thereby occasioned gives rise to vibrations

* The tension may be more accurately estimated by means of the sphygmomanometer of Von Basch, which will be hereafter described.

† *Loc. cit.* ‡ *Lehrb. der Untersuchungs Methode.*

in the blood stream, and to an audible murmur coincident with the arterial expansion. If the pressure be increased, this murmur passes into a sharp sound.

2. *In Disease.*—Sounds or murmurs may be heard in the arteries under three pathological conditions:

(*a.*) *Murmurs conducted from the Heart.*—It is, as a rule, aortic murmurs (both systolic and diastolic) which are propagated into the arteries, although mitral murmurs are occasionally to be heard very faintly in the carotids.

(*b.*) *Sounds and Murmurs originating in the Arteries in consequence of general Circulatory Disease.*—In aortic incompetence a sound coinciding with the arterial expansion may be heard in all the accessible arteries of the body, due almost certainly to the rapid transition from extreme relaxation to extreme tension which the arterial coats then undergo. A double sound over the femoral artery is also sometimes to be heard in such cases, as was first pointed out by Conrad,* and subsequently more fully studied by Duroziez,† Traube,‡ Friedreich,§ and others, and lately by Senator.‖ The first of these sounds, that coinciding with the arterial expansion, originates in the arterial coats, as already described; and the second arises, in the majority of cases, not in the artery, but in the femoral vein, as a result of coexisting tricuspid incompetence. Very rarely, indeed, cases occur in which later sounds are of arterial origin (the tricuspid valve being intact), and these result from aortic incompetence.

A double murmur may be produced in the femoral artery in cases of aortic incompetence by pressure with the stethoscope, the one murmur being caused by the pulse wave, the other by the returning backward wave, which in such cases flows towards the heart during the arterial collapse. This double murmur may also occasionally be heard in cases of anæmia, typhoid fever, etc.

(*c.*) *Murmurs originating in the Arteries in consequence of Local Changes.*—Such murmurs are to be heard over aneurisms and vascular tumors, but more important are the subclavian murmurs. While occasionally occurring in healthy persons, murmurs over the subclavian arteries are much

---

* *Zur Lehre über die Auskultation der Gefässe.* Giessen, 1860.
† *Arch. gén. de méd.*, 1861.
‡ *Vide Berl. kl. Woch*, 1867, No. 44.
§ *Deutsches Arch. für kl. Med.*, xxi. p. 205., and xxix., 1881.
‖ *Zeitschr. f. kl. Med.*, iii., 1881.

more frequently heard in cases of phthisis, due probably to adhesions between the pleura and the vascular walls, and hence much influenced by the respiratory movements.

The encephalic murmur which Fisher discovered in children has, so far as our present knowledge goes, no diagnostic significance.

The cephalic murmur which Tripier has recently shown to be present in anæmia over the mastoid process, the occiput, and the eyeball, is supposed by him to be of arterial origin. It is coincident with the expansion of the arteries; but it is a little difficult to see why, if it be an arterial murmur, it should be loudest in these three positions, and hence Gibson has referred it to vibrations in the internal jugular vein produced by the systolic stroke of the neighboring carotid artery, and conducted into the venous sinus. This ingenious explanation does not, however, seem to me satisfactory. I am inclined to regard this cephalic murmur as venous, but as the product of the systolic augmentation of the venous current which takes place within the skull, and within the eyeball, due to the fact that in each case we are dealing with a closed box, which is practically incapable of expansion. Hence, when a sudden increase of arterial blood takes place in them, when the arteries are distended, an equally sudden outflow must occur through the veins. To the vibrations in this sudden venous current I am inclined to ascribe this very interesting cephalic murmur. The watery condition of the anæmic blood is, of course, the important factor.

Capillaries.

The state of the capillary vessels need not be specially noticed here, seeing that the more noteworthy points have been elsewhere discussed.

Veins.

Knowledge concerning the condition of the veins may be obtained by inspection and by auscultation. Palpation by the fingers, and percussion, are not fitted materially to aid the physician.

Inspection.—By inspecting the veins we ascertain, firstly, their state as to fulness, and secondly, whether the blood contained in them undulates or pulsates.

Overfilling of the veins results either from local obstruction, when the vein becomes tense on the distal side, and such of the collateral branches as are not compressed enlarge so as to carry on the circulation, or from interference with the venous circulation generally. Examples of the variety of engorgement arising from local obstruction are to be found in cases of thrombosis of any of the larger venous trunks, or where the pressure of an aneurism or other mediastinal tumor gives rise to overfilling of the veins of the arm. The distension of the cervical veins which arises where the general circulation is interfered with has already been described.

Undulation of the Veins of the Neck.—The pulsations in the cervical veins which correspond to the movements of the heart have been already remarked upon. It only remains to mention the undulation which the respiratory movements sometimes produce in the jugular veins. When the cervical veins are overfilled as a result of pulmonary emphysema, or of mitral stenosis, each inspiration diminishes the venous distension, while each expiration increases it, and so the veins show a constant undulation.

Auscultation.—Although in cases of tricuspid incompetence systolic sounds are occasionally to be heard over the jugular and femoral veins, the only auscultatory sign which here demands attention is the humming murmur, the so-called *bruit de diable* which is very frequently to be heard in chlorotic females over the bulb or dilatation of the internal jugular vein, and more rarely over the large intrathoracic venous trunks, the superior vena cava, and the innominatic veins. Venous murmurs in the former are best heard at the right border of the sternum, from the first right intercostal space to the third costal cartilage. The murmur in the right innominate vein is usually loudest at the sternal end of the first right costal cartilage, and that in the left over the manubrium sterni. Occasionally a venous hum is to be heard in dilated thyroid veins, and in the subclavians, axillary, brachial, and femoral veins. In venous auscultation, it must be borne in mind that the slightest unnecessary pressure with the stethoscope may develop an artificial murmur.

The *bruit de diable*, as met with in the jugular vein (generally loudest on the right side), is usually of a continuous soft humming character, and occurs very frequently in

health. Winterich * detected it in 80 *per cent* of the Bavarian cuirassiers whom he examined. Only when the murmur is strong and loud is it pathological, or can it be taken as evidence of the existence of anæmia; and we may, with Friedreich,† define the pathological venous murmur as limited to those cases in which a thrill is perceived when the finger is applied over the jugular bulb, or in which the murmur is sufficiently loud to be heard when the ear is removed a little way from the stethoscope, or to become apparent to the patient himself, and finally, when a murmur can be perceived over the intrathoracic venous trunks.

These venous murmurs appear to depend for their production upon three factors—1st, upon the rapidity of the blood current; 2d, upon the change in the calibre of the vein at any particular point (such as occurs in a marked manner at the jugular bulb); and 3d, upon alteration in the quality of the blood, whether this consists in an actual or only a relative increase of the watery elements.

Usually the jugular humming murmurs are continuous, but they very often vary in intensity, and occasionally are actually intermittent. They are influenced in the following ways:

1. *Changes in the Posture of the Patient.*—When the head is turned to the opposite side the murmur becomes much intensified, owing to the compression of the vein by the muscles and fascia. Even when no murmur exists when the head is held straight, a faint bruit may be developed when the head is rotated; especially if firm pressure be made with the stethoscope in addition.

Owing to the acceleration of the blood flow in the veins the murmur is louder when the patient sits or stands than in the recumbent posture.

2. *The Movements of Respiration.*—Sometimes the venous murmur in the jugular is only audible during deep inspiration, and if it be continuous it is almost invariably intensified by that action, in both cases, for this reason—viz., that during inspiration the flow of blood in the vein is accelerated. The same usually holds good with regard to murmurs in the femoral vein, although in rare instances the reverse obtains, and we meet with the remarkable phenom-

---

* *Deutsche Klinik*, 1850.

† *Deutsch. Arch. f. kl. Med.*, vol. xxix. (1881) p. 263.

enon of a femoral murmur which is expiratory in rhythm,* this probably resulting from the increased abdominal pressure which the descent of the diaphragm occasions, and which retards the blood current in the femoral vein.

3. *The Movements of the Heart.*—The anæmic murmur in the jugular vein is sometimes diastolic in rhythm, as was first pointed out by Chauveau,† who ascribed it to the increased blood current in the vein which is the result of the diminution of pressure in the superior vena cava produced during diastole, and which stands closely related to the negative diastolic pressure in the ventricle. While this is no doubt one cause of this diastolic venous hum, it appears to the author extremely probable that the cause suggested by Friedreich is likewise operative—viz., that the pulsations of the aorta compress the superior vena cava during the cardiac systole, thus allowing an uninterrupted flow of venous blood during diastole.

CHAPTER XIII.

CIRCULATORY SYSTEM—(*Continued*).

GRAPHIC CLINICAL METHODS.

WHEN Chauveau and Marey first introduced to the notice of the profession the sphygmograph and cardiograph, it was hoped that a new and more accurate examination of the heart and circulatory system would soon replace the former methods. This hope has not been realized. There is, indeed, little difficulty in obtaining tracings of the pulse wave and heart beat, and these tracings, moreover, are found to vary greatly in different diseases; but the true meaning of these differences is as yet by no means thoroughly understood. The reason for this lies partly in the fact that the meaning of the normal pulse and heart curve has not yet been explained, in all its details, in a fully satisfactory manner. Still, even now, certain trustworthy facts can be obtained by the use of the recording instruments referred to, and the number of these facts will necessarily increase as the characteristics of the normal pulse wave and

---

* Friedreich, *loc. cit.* † *Gaz. Méd. de Paris*, 1858.

heart beat and the modifications which they may undergo in health become more fully understood. Moreover, the permanence of the records which may be obtained by the use of such instruments, their value in illustrating the history of individual cases, together with the fact that these instruments give results which are more purely objective than those obtainable by other methods, amply justify a somewhat full description of the manner of using the sphygmograph and cardiograph, together with some account of the results obtainable by their help.

Sphygmograph.—The original instrument of Chauveau and Marey which, since its introduction, has been repeatedly modified in detail by Marey himself and by others, in its present form (as supplied by Breguet) is the most favorite form of instrument employed at the present day. Some have sought to introduce the so-called transmission sphygmograph, but it seems to be generally accepted that the advantages which this form of instrument presents in certain particulars are more than counterbalanced by very obvious defects. All of these transmission sphygmographs are similar in principle, consisting of two closed Marey's tambours—one being influenced through the medium of a button pressing on the radial artery, while the second tambour, joined to its fellow by an india-rubber tube, is arranged to move a recording lever which writes on the blackened surface of a revolving cylinder. The great advantage presented by this form of sphygmograph is that by its means we can obtain curves of practically indefinite length, more especially if the revolving cylinder move round a spiral spindle. On the other hand, these instruments are all more or less cumbersome and expensive, while the introduction of a long column of air to transmit the form of the pulse wave to the recording lever introduces many serious possible errors. However, for investigation of certain special points, as, for example, where simultaneous tracings of the pulse, heart, and respiratory movements are desired, the transmission sphygmograph is the only instrument which can be employed. Of these Marey's polygraph * is probably the most perfect and convenient.

On account of its comparatively small cost and convenient size, it is probable that the original Chauveau and

* *Vide* Marey, *La Circulation du sang à l'état physiologique et dans les Maladies.* Paris, 1881.

Marey's sphygmograph, in its most recent modification, will still continue, at least for some time, to be the most commonly used instrument. The principle of its construction is to be found in all text-books of physiology, and need not therefore be dealt with here; and I will confine myself to a description of the typical pulse curve, and the modifications which it undergoes in health and in disease.

The typical healthy curve, of which the accompanying tracing (Fig. 1) is an example, is usually divided into an ascending and descending portion, either or both of which may present certain secondary undulations. In its most typical form (as in Fig. 1) the ascending line (*a* to *b*) rises abruptly at first, and afterwards more slowly, till it reaches its highest point. Then descending more obliquely, it usually presents a more or less well-defined notch or indenture (*c*) before it reaches the principal notch or valley (*d*). This latter notch is best known as the dicrotic notch, and is of great importance, corresponding as it does exactly to the closure of the aortic valves. After the dicrotic notch, the curve describes a slight elevation before descending to its lowest level, in the course of which descent a low wave-like eminence (*f*) is not unfrequently to be discovered. Since the point (*a*) corresponds to the opening of the aortic valves, and the point (*d*) to their closure, the artery is, during the time represented by the interval between these two lines, in free communication with the interior of the ventricle, while, during the time of the rest of the curve the artery is cut off from the heart.† The point *d*, therefore,

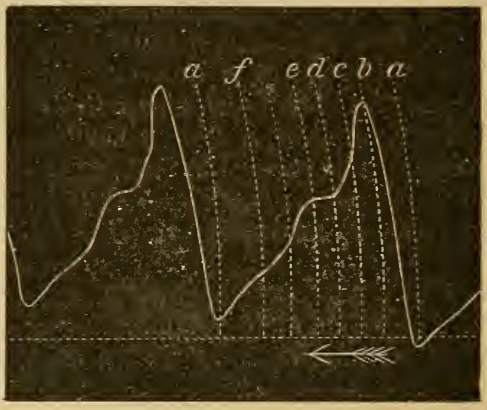

FIG. 1.—Normal Pulse Curve.*

* For this curve and those which follow, as well as for much help and advice in the preparation of this chapter, I am indebted to the kindness of my friend Dr. C. S. Roy, of the Brown Institute, whose surpassing skill in instrumentation is well known.

† The whole pulse wave is delayed in its transmission from the commencement of the aorta to the radial artery, but the delay of the different parts of the curve is usually tolerably equal, so that the relative distance between the up-stroke and the dicrotic notch remains the same.

forms the most natural division of the pulse wave into its more fundamental parts, the causes which influence its form during the first half being essentially different from those which modify the second half. Keeping this fact in view, and premising that it is almost always easy to find in any curves, of whatever form the point which corresponds to *d* in our typical curve, I now proceed to describe the modifications of the pulse wave which are to be met with.

And, first, with regard to changes in the first half of the curve.

Anacrotic Pulse.—The typical pulse curve, of which I have given an example above, is not infrequently called dicrotic, owing to the fact that it presents a fairly well-marked notch in its descending part, although some authors prefer to restrict the term dicrotism of the pulse wave to cases in which that notch is abnormally well marked. In contradistinction to the dicrotic pulse, it is the custom to call those pulse waves in which a more or less well-marked notch occurs in the ascending line as anacrotic. The tracing (Fig. 2) annexed shows a fairly typical example of the anacrotic pulse wave. It can be seen that it differs from the dicrotic or normal pulse-wave only in the part which lies between the lines *a* and *d;* in other words, in that part of the pulse wave which corresponds to the time when the aortic valve is open. We would, therefore, expect, *à priori*, that this change in the form of that part of the pulse wave must be due to some difference in the relation between the quantity of blood thrown out of the left ventricle and the elastic resistance offered by the aorta and larger arteries. Let us suppose that the arteries are relatively lax, and that the quantity of blood thrown out of the ventricle is not above normal, then it is not difficult to understand that the ventricle will more readily and more rapidly empty itself than when the vessels are relatively rigid. The result of this is that the point of the pulse wave, where the highest pressure exists, and which corresponds to the highest point of the pulse curve, will occur nearer its commencement than would otherwise

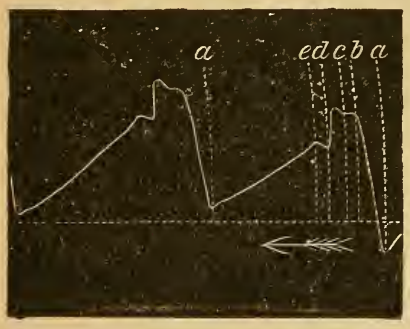

Fig. 2.—Anacrotic Pulse Wave.

be the case. Let us, on the other hand, suppose an extreme case, in which the arteries are very rigid, as in well-marked atheroma, or calcification of the larger vessels, these latter, as the contents of the ventricle are forced into them during systole, do not expand to receive the contents of the ventricle, but act more like rigid tubes, the result of which is that during the cardiac systole the inflow into the vessels, which is always greater than the outflow at that period, produces a continuous rise in arterial pressure during the whole time of systole. The point of highest pressure of pulse wave, or, in other words, the highest part of the pulse tracing, is thus thrown toward the end of the ventricular part of the pulse curve, or, in other words, closer to the dicrotic notch, *d*, which marks the end of the systole. In other words, in cases where the larger arteries are not fitted to contain the quantity of blood contained in the ventricle, the latter forces the blood at first against a comparatively weak resistance, which, however, goes on increasing very rapidly as the large arteries become gradually more and more tensely filled; and the pressure within these latter necessarily rises from the commencement to the end of the cardiac systole. This, then, is the reason why, in such circumstances, the highest part of the pulse curve is nearest our line *d*, or the dicrotic notch which corresponds to the end of the ventricular systole.

I have as yet said nothing of the indenture (*c*) which precedes the dicrotic notch, and which, on that account, is usually described as the pre-dicrotic notch. The exact significance of this notch is still by no means so fully understood as is desirable. It would seem that its appearance results from the fact that, at the moment when the aortic valves are forced open, the column of blood contained in the aortic arch and larger branches receives a sudden impulse towards the periphery, and the inertia of this column of blood, thus set in comparatively rapid motion, produces a negative wave at the commencement of the aorta, which is propagated towards the periphery in the same manner as the positive wave which preceded it. I have spoken of the causes which may theoretically produce anacrotism, and also the probable cause of the pre-dicrotic notch, and must now proceed to refer to the conditions under which, in practice at the bedside, we find the ventricular part of the pulse wave so modified.

If the glottis be closed, and the pressure within the thorax

and abdomen be raised by powerful continuous contraction of the respiratory muscles, we produce a change in the distribution of the blood in the arteries and veins. The intra-abdominal and intra-thoracic veins are relatively empty, and an abnormally large quantity of blood accumulates in the systemic arteries. During this state the arterial walls are more or less powerfully distended, and, following known laws regarding arterial elasticity, they are in that condition more rigid than when their calibre is normal. Even in tolerably young subjects, by this means we can easily produce artificially an anacrotic pulse wave, the arteries being rendered relatively rigid in relation to the quantity of blood which is forced into them at each ventricular contraction. This arterial engorgement or high pressure, only temporary in such an experiment, is, however, lasting in certain diseased conditions, the most marked of these being the arterial high pressure which accompanies certain forms of chronic kidney disease, in which latter case the conditions are still more favorable for the production of an anacrotic pulse wave, seeing that not only are the arteries abnormally rigid from the distension, but also that the quantity of blood forced into them with each contraction of the ventricle is relatively and absolutely great, owing to the existence of eccentric hypertrophy of the left ventricle. Analogous conditions occur, as already indicated, in cases of atheroma or calcification of the larger arteries, such as occur in old age. The conditions, therefore, which produce the anacrotic pulse wave are in practice either abnormal distension of the larger arteries, accompanied or not by hypertrophy of the ventricle, or rigidity of the arterial walls due to changes in molecular structure of their middle coats. The more marked these conditions are, the more is the second elevation (*c*) higher than the first. (*b* in Figs. 1 and 2.) In practice, all imaginable intermediate forms between the typical pulse wave of health and the typical anacrotic pulse wave, as in Fig. 2, are encountered, and it is usually easy in each individual case to tell from the other phenomena whether the anacrotism be due to simple distension of the arteries from high pressure, to molecular change in the arterial coats, or to hypertrophy of the heart. From what I have said it will be understood that although the anacrotic pulse wave very often means an abnormally high arterial pressure, this is by no means always the case. Finally, before leaving the changes confined chiefly or en-

tirely to what I have named the ventricular part of the pulse wave, a word may be said regarding the conditions which favor the appearance of a well-marked pre-diastolic notch. The condition fitted to produce this notch in its most marked form is that in which the part of the systemic arteries nearest the heart is abnormally rigid; for it need scarcely be said that if this latter part of the systemic arterial system is fairly elastic, it will contract behind the suddenly impelled first wave, and prevent more or less completely the formation of a negative wave or tendency towards a vacuum at the commencement of the aorta.

We now turn to consider a different series of changes in the form of the pulse wave, which are due to changes in the arterial circulation of an entirely different kind from those above referred to, and in which practically invariably the dicrotic notch is abnormally exaggerated.

Abnormally Dicrotic Pulse Waves.—I have said above that on closing the glottis and contracting powerfully the respiratory muscles, the systemic arteries are at first abnormally filled with blood; this abnormal distension very soon, however, gives place to an abnormal emptiness of these vessels, owing to the fact that the pressure on the intrathoracic veins diminishes the quantity of blood which reaches the ventricle, the result being that the blood accumulates chiefly in the veins of the head and limbs. The artificial arterial anæmia so produced leads to a characteristic change in the form of the pulse wave, which becomes, as in Fig. 3, smaller in size and more markedly dicrotic than even the normal pulse, while all trace of anacrotism completely disappears. It is unnecessary for us to go minutely into the theory of the production of the abnormally dicrotic pulse wave. For practical purposes it will suffice to refer to the conditions which lead to the appearance of this form of curve. Roughly speaking, these may be said to consist in abnormal emptiness of the arterial system, such as is produced, for example (*a*), by anæmia after venesection, in which case the absolute quantity of blood in the arteries is diminished, although these latter contain relatively normal amount; (*b*) in cases of unusual expansion of the arterioles and capillaries leading to a relatively rapid outflow from

FIG. 3.—Abnormally Dicrotic Pulse Wave.

the arteries, as in the condition produced by amyl-nitrite inhalation; or finally, dicrotism may be produced by (*c*) diminution in the quantity of blood which enters the aorta through the ventricle—the most marked examples of which are to be found in cases of uncompensated mitral regurgitation.* Such are the conditions which, in practice, are found to produce the dicrotic pulse wave; and it may be noted in passing that simple or pure dicrotic pulse wave invariably results from abnormally low arterial pressure, the cause of which, in individual cases, it is rarely difficult to discover.

Hyperdicrotic is the term applied to that form of the dicrotic pulse in which the dicrotic notch descends lower than the commencement of the systolic rise. This is due to the fact that each successive cardiac systole follows its predecessor before the pressure within the artery has fallen below that which it presented at the dicrotic notch. This form of curve (Fig. 4), although presenting a notch in its ascending part, is due to entirely different conditions from those which produce the true anacrotic pulse wave, with which it can never in practice be confounded, owing to the fact that the rounded smooth eminences of which it is made up shows it at a glance to be of the dicrotic type; it is, in fact, an exaggerated dicrotic pulse-wave.

Fig. 4.—Hyperdicrotic Pulse Wave.

The anacrotic and the dicrotic pulse waves are the two principal simple modifications which are met with, but there are various intermediate or combined pulse waves due to combination of the conditions fitted to produce the anacrotic and the dicrotic waves—for example, in cases of aortic regurgitation with hypertrophied ventricle, the first or ventricular part of the curve usually is of the true anacrotic type, while, on cessation of the systole, the reflux into the heart causes more or less powerful negative wave producing an abnormally deep dicrotic notch. In addition to this, the rapid filling of the comparatively empty arteries with each

* Not unfrequently two or more of these causes may be combined, as happens in fevers.

ventriculatory systole leads to an abnormally steep and high ascending limb of the curve.

We have given the conditions which lead to the two principal forms of pulse wave met with in disease; but, as I have said, all possible combinations of these conditions are constantly occurring, leading to some less well-defined type of pulse curve. Into a detailed account of these more complicated pulse curves we cannot enter here. What we have already said will enable the observer to understand the meaning of each. Examples of these are found in the various modifications of pulse curve in prolonged fever cases, in the early stages of which it is often high, bounding with a tendency towards the anacrotic type, but gradually from day to day becoming more and more dicrotic, and not unfrequently being eventually hyperdicrotic.

Cardiograph.—The results which may be obtained by the use of the cardiograph are on the whole less satisfactory than those of the sphygmograph. This is in part due to the fact that none of the instruments at present in use for recording the contractions of the human heart can compare with the sphygmograph, in so far as compactness and accuracy are concerned. While some observers, such as Landois, have attempted to record the form of the apex beat by applying to the surface of the chest the sphygmograph of Marey, the majority of the instruments employed at the present day are transmission instruments, being constructed in the same manner as the transmission sphygmograph, which I have already described. It is extremely desirable that we should have at our disposal some direct acting cardiograph similar in principle to the sphymograph of Chauveau and Marey. Such, however, has not as yet been described. In the meantime the transmission cardiograph is the one which is almost universally employed. That of Burdon Sanderson,* or the polygraph of Marey,† are amongst the best, if not the very best. I need not enter upon the manner of using these and similar instruments, but will proceed to describe, firstly, the typical normal heart curve; and secondly, the principal modifications which it may present.

Normal Heart Curve.—In Fig. 5 is represented a typical

---

* "Handbook of the Physiological Laboratory," edited by Burdon Sanderson.

† *Loc cit.*

normal curve of this kind. The curve, it will be seen, immediately after rising from its lowest point (*f*), describes a more or less well marked rounded elevation between *f* and *a*, and from *a* it ascends at first rapidly, afterwards somewhat more slowly, to its highest point *b*, from whence it describes a more or less obliquely descending, usually undulating line to *e*, after which the curve descends, at first slowly, then more rapidly, and finally with increasing slowness, until the point *f* is reached. That part of the curve lying between *f* and *a* corresponds in time to the con-

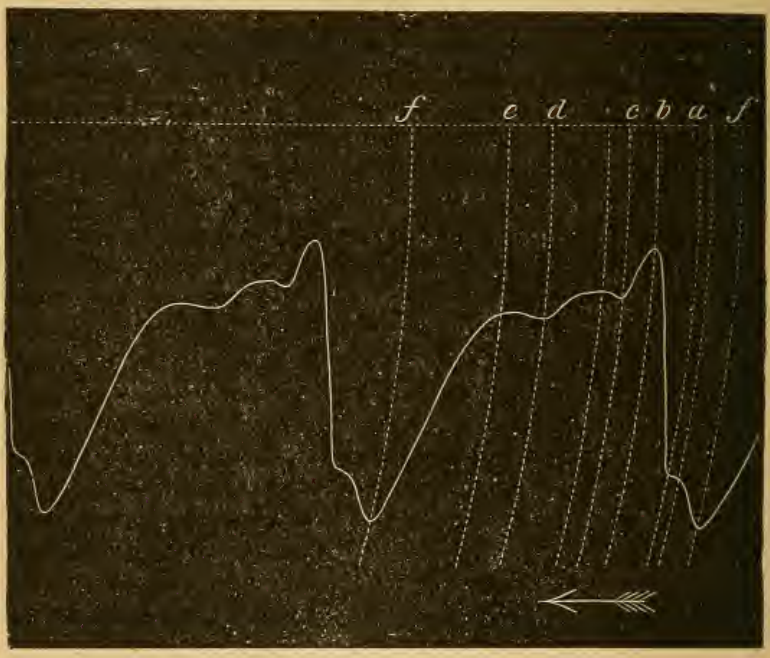

FIG. 5.—Normal Heart Curve.

traction of the auricles, and when the curve is taken from the apex, the elevation between *f* and *a* is due to the more or less sudden filling of the ventricles which results from the auricular contractions. That part of the curve lying between the lines *a* and *e* is produced during the time of contraction of the ventricular muscle, while the part from *e* to *f* corresponds with the passive expansion of the ventricular muscle. In so far as the ventricles are concerned, we may divide the whole heart curve into two parts—viz., first, that from *a* to *e*, during which the ventricular muscle is in a state of contraction; and second, that from *e* to *a*, which corre-

ponds to the ventricular diastole. The sudden rise from *a* to *b* is produced by the tightening of the ventricles over their contents, and the point *b* corresponds in time to two important phases of each heart beat—viz., first, the moment of closure of the auriculo-ventricular valves; and secondly, the moment when the heart muscle has fairly grasped its contents. The height of *b* over *e* gives some indication of the difference in antero-posterior diameter of the heart at commencing systole as compared with the end of the systole, for it need scarcely be said that the larger the quantity of blood contained in the ventricle at the commencement of the ventricular systole, the greater will be its antero-posterior diameter, and therefore the more powerful impulse will be given to the chest wall and cardiograph button. As the heart empties itself during systole the antero-posterior diameter of the ventricles diminishes with corresponding rapidity, and the pressure against the chest wall and cardiograph button falls in the same ratio. The result of this is that, *cæteris paribus*, the degree to which the line joining *b* and *e* descends gives a valuable indication regarding the quantity of blood thrown out by the ventricles at each systole. The meaning of the notches *c* and *d* is not satisfactorily understood. This much, however, is certain—viz., that they are not due to inertia vibrations of the recording lever, as has been asserted by some, and also that in not a few cases the form of the curve lying between the lines *a* and *d* of the cardiogram resembles very closely that lying between the lines *a* and *d* of our normal pulse curve (Fig. 1.) In other words they are probably due to oscillations of the column of blood contained in the ventricles and larger arteries. The notch *d*, when well marked, corresponds to the conclusion of the outflow of blood from the heart, and is therefore the analogue of the dicrotic notch of the pulse wave. It must be added, however, that it is by no means uniformly to be seen. Of greater importance is the position of the last elevation or corner of the curve at *e*, which can in almost all curves clearly be made out. This elevation marks the commencing relaxation of the ventricular muscle, and by measuring the distance between the lines *a* and *e* in the manner which will be described in a note appended to this chapter, we are enabled to learn with absolute accuracy the duration of the ventricular systole in any given case. I must mention, in passing, that the duration of the ventricular systole, and the duration of the

outflow from these cavities, by no means necessarily or even usually correspond. The ventricular muscle contracts with a certain definite force, and remains contracted for a certain definite time, neither of these being influenced by the quantity of blood contained in the ventricle at the commencement of its contraction. The result of this is, that where a very small quantity of blood is contained in the ventricles at the commencement of their contraction, the outflow from them may have concluded some tenths of a second before the ventricles begin to relax. The distance between the lines *e* and *f* gives some indication of the rapidity with which the heart muscle has relaxed after the conclusion of its contraction. Where the elasticity of the heart muscle is modified, as when the blood contains a largely diminished quantity of oxygen, the ventricular muscle takes a longer time to relax than is normally the case, and the curve from such a beat descends less rapidly than in health.

The cardiographic curve then enables us to measure with very considerable accuracy the absolute and relative duration of the different phases of the cardiac revolution. It also gives us some idea of the force of the ventricular contraction corresponding to the height of the line *a* to *b*, and it further affords valuable information regarding changes in the force and frequency of the heart's action which make up the different forms of irregularity of the heart.

It is unnecessary to refer more in detail to the normal typical heart curve, and I turn now to mention those diseased conditions which modify its form; and first, with regard to the cardiogram in aortic regurgitation. After what has been said regarding the meaning of the various parts of the normal heart curve, it is not difficult to understand in what way these may be modified in a typical case of aortic regurgitation. In the first place, the ventricle, before the contraction of the auricles, is abnormally distended with blood; and on the auricles propelling their contents into the already filled ventricle, an abnormally great distension of the ventricles occurs. The result of this is that, in cases where there is no failure in the power of the auricular walls, the elevation between the letters *f* and *a* is abnormally high. On ventricular contraction occurring, the antero-posterior diameter of the heart diminishes very rapidly, corresponding with the abnormally large quantity of blood contained in the ventricle, so that the line joining points corresponding to *a* and *e* is unusually steep, while

the regurgitation of blood through the incompetent aortic valves, after the cessation of the systole, causes a dilatation of the relaxing cardiac muscle sufficient to produce in most cases a very well-marked rise after *e*. It is important to note that, in the heart-curve of well marked aortic regurgitation, it is often impossible to find the exact point corresponding to *e* in the normal curve at which the systole suddenly ceases. The corner of the curve preceding the descent is usually in the aortic regurgitation cardiogram some fraction of a second later that the time of commencing relaxation. In all, or nearly all, cases of aortic regurgitation, the heart curve presents two well-marked peaks, and this may be said to be the distinguishing character of the cardiogram of that disease, and, roughly speaking, the more marked this bicornual character is, the greater is the incompetence of the valve. Such is the curve when the ventricular muscle is comparatively unimpaired in contracting power, as, for example, in sudden rupture of one of the cusps of the valve, or when one of these is artificially destroyed in the lower animals; but where the ventricle no longer completely empties its contents at every contraction, the fall of the line from *b* to somewhere about *e* becomes, as we might anticipate, less and less steep, due, it need scarcely be said, to the slighter diminution in the antero-posterior diameter of the ventricles, which occurs when the ventricle no longer empties itself completely at each contraction. In these cases then, the bicornual character is not so well marked as is otherwise the case in that disease, but still it is usually sufficiently recognizable.

With regard to the curve in cases of mitral incompetence, we would anticipate, where this condition was well marked, that the heart curve would be modified chiefly, if not exclusively, at that part which corresponds in time to the closure of the auriculo-ventricular valves; in other words, at the point marked *b* in Fig. 5; and this to a certain extent is the case. As a rule, we find the ascending line from *a* to *b* less steep than is normally the case, and the peak at *b* rounded off to some extent This is apparently the only characteristic change in the form of the heart curve which results from simple mitral incompetence; but it is by no means usually well marked, owing to the fact that the large quantity of blood which leaves the ventricle during systole causes a very considerable diminution in the antero-posterior diameter during ventricular systole; so that

the line from *b* to *e* is unusually steep, thus tending to cover the rounding off which the peak *e* would otherwise present. It is rare to fine that the reflux of blood from the auricle into the ventricle which follows the conclusion of the systole of the ventricle distends the latter with sufficient force to cause, as is the case in aortic regurgitation, second elevation after *e*. Finally, where the auricles are hypertrophied, the auricular elevation in the heart-curve of mitral incompetence is abnormally well marked. Such are the changes produced by simple mitral incompetence, which has been more or less completely compensated by hypertrophy of the auricle and dilation and hypertrophy of the ventricle; but in cases where either the auricle or the ventricle begins to fail, there are endless modifications, which what has above been said regarding the meaning of the different parts of the normal heart curve will enable the physician readily to understand; but it should be added that in advanced mitral cases, where irregularity is a pronounced element of the case, the heart curve becomes so. *bizarre* in form that it is difficult and sometimes impossible to understand what is the meaning of the different elevations which it presents.

With regard to pure mitral stenosis, we would *à priori* expect that the ascending line from *a* to *b* would be abnormally steep, owing to the abnormal rigidity of the mitral valve, and that the peak *b* would be unusually sharp, corresponding as this does with the thump which is characteristic of the disease in question, and such is certainly sometimes the case; but when it is remembered how exceedingly rare is stenosis of the mitral valve uncomplicated with regurgigation, it need cause no wonder to find that the cardiogram in mitral stenosis is by no means characteristic or typical. Still, the curve in all, or nearly all, cases presents sufficiently well-marked deviations from the normal which are fitted to throw much light upon the condition of the heart in individual instances; and careful attention to the period of time in the heart revolution at which these abnormalities take place readily indicates their meaning.

There are many modifications of the heart curve which it is impossible to describe here in detail; and, indeed, in practice, almost every imaginable combination of the abnormalities above described is encountered.

Sphygmomanometer.—Very recently, Professor S. von

Basch has introduced* an instrument, to which he gives the above name, for the purpose of estimating the blood-pressure in the human subject, and which has undoubtedly considerable clinical value. In principle it closely resembles an apparatus previously described by Prof. C. S. Roy and myself,† which was more specially adapted for estimating the blood-pressure in the lower animals. In Von Basch's sphygmometer, a small cushion of membrane is made to press upon the skin over the radial artery, and the pressure is communicated through water to a column of mercury, by which its value can be ascertained. The pressure of the cushion upon the radial artery is gradually increased, until all pulsation in the vessel beyond the constricted point ceases; and this point is taken as the maximum arterial pressure.

While open to many sources of error, the readings of this instrument, if taken with sufficient care, appear to give results which are approximately accurate; at any rate, quite sufficiently so for ordinary clinical work. In healthy men between the ages of twenty and sixty the pressure averages about 150 mm., but may be as low as 135 mm., or as high as 170 mm. High pressures much exceeding these, and running as high as 245 mm., are met with in such affections as chronic Bright's disease with cardiac hypertrophy, while in such diseases as anæmia, phthisis, etc., the blood-pressure is found to be very low.

This instrument seems to be specially suited for watching the progress of individual cases from day to day, and in particular for observing the effects of treatment upon the blood-pressure.

Note on the Measurement of Tracings.

In order to measure the relative duration of different parts of the pulse wave (and the remarks which follow apply equally to heart curves), a certain method is necessary to correct the error, due to the fact that the point of the lever describes, not a straight vertical line, but the segment of a circle. The most convenient method of doing this is as follows: Having obtained a satisfactory curve

---

**Zeitschrift f. kl. med.*, vol. ii., 1881, p. 79; and vol. iii. p. 502.

†*Verhandlung der physiol. Gesellschaft zu Berlin*, 15th Feb., 1878. *Journal of Pysiology*, vol. ii. p. 323.

from the radial artery, the sphygmograph is removed from the arm, and the paper in its holder is again passed through the clockwork, the point of the lever being fixed so as to draw a straight abscissa—*i. e.*, one parallel to the line of movement of the paper. This line may be drawn either below the curve or through it; but where absolute accuracy is desired, it ought to be at the level of the centre of that circle of which the lever describes a segment when the paper is at rest. The paper is then either drawn backwards or put a third time through the clockwork, and successive parallel curved lines are described by the lever point connecting those portions of the curve which it is desired to measure with the abscissa line. In Fig. 1 there are certain dotted lines, of which the horizontal represents the abscissa line, and the vertical curved lines represent segments of the circle described by the recording lever. Since the movement of the clockwork of the sphygmograph is, during the time when any single pulse curve is being recorded, fairly uniform, and since the rapidity of the heart beat is readily learned by the watch, it is not difficult to say what fraction of a second corresponds to the distance between any two of our vertical curved lines. For example, in Fig. 1 the pulse was, let us say, sixty in the minute; and as the distance from the vertical lines *a* to *c* is roughly one-third of the whole, we know that the duration of that portion of the pulse wave is equal to one-third of a second. In the same way, the duration of any other portion of the pulse wave may be determined with sufficient accuracy. This method enables us to learn the *duration* of any of the phases of the pulse curve, while the relative importance of the various parts of the same curve—that is, the tension of the blood in the artery at different points—is represented by the height of the tracing as measured from a horizontal line running through the lowest part of the pulse wave.

CHAPTER XIII.

Respiratory System.

The first symptoms which require notice in connection with the respiratory system are:

Subjective Phenomena, such as pain, tickling, burning, etc., which are frequently felt over the larynx, trachea, and bronchi, when these structures are the seat of disease, and are usually aggravated by pressure, and by the acts of speaking and coughing. *Pain* may manifest itself in connection with disease of the lung tissue, but it attains its greatest importance in cases of pleurisy, where the pain has a peculiar dragging, shooting character, is increased by pressure, and by any movement of the thorax. Its differential diagnosis is very important.

Distinguish the pain of pleurisy:

(1.) *From the pain of pleurodynia*, or rheumatism of the intercostal muscles. In this condition the pain comes on with excessive suddenness, after some abrupt movement, and is unaccompanied by pyrexia or by friction sound.*

(2.) *From the pain of intercostal neuralgia.*—In this affection there are commonly three tender points in the course of the affected nerve, one close to the vertebral column, one in the axilla, and a third over the terminal branches near the sternal border. The presence of these points, the neuralgic character of the pain, and the absence of all pulmonary physical signs, except such alteration of the respiration as the pain occasions, will suffice to distinguish this affection from pleurisy.

(3.) *From the pain of Herpes Zoster.*—This eruption, which follows the course of the intercostal nerves, is sometimes preceded by severe pain, usually of a burning character. The marked cutaneous hyperæsthesia which frequently accompanies this pain will suffice to distinguish it from pleu-

* It must be remembered, however, that this auscultatory phenomenon may be awanting in the early stage of pleurisy, so that the physician may have to refrain from a positive diagnosis until this symptom has had time to develop.

risy. The eruption is often followed by long persisting neuralgia, the nature of which, however, will be at once made clear by the history of preceding herpetic eruption.

(4.) *From the Pain of Periostitis and other Surgical Affections of the Thoracic Wall.*—Careful examination of the ribs will make clear the nature of such pain.

Breathing will be more conveniently considered hereafter.

Cough.—The removal of foreign substances from the respiratory passages is effected by means of the acts of sneezing and coughing—two forms of explosive expiration which are both, as a rule, excited reflexly, and which both consist in a closure of the respiratory passages after a deep inspiration, followed by a sudden, forcible, and noisy opening of the same, the result of a powerful expiratory effort. In the case of sneezing, the closure is effected by the pressure of the soft palate by means of the tongue, on the posterior wall of the pharynx, while in coughing the closure takes place at the glottis. Of these two acts that of coughing is by far the most important. Sneezing need not be further considered here.

Coughing is excited by iritation of the terminal branches of the superior laryngeal nerve distributed to the mucous membrane of the larynx and trachea. The inhalation of cold air, or of air laden with dust, the passage into the larynx of particles of food, or other foreign bodies, and the collection of secretions, or of such morbid products as blood or pus, all tend to excite coughing, which is more liable to occur when in addition there is hyperæsthesia of the parts, the result of catarrh or inflammation. The terminal branches of the vagus distributed to the bronchi, lung tissue, pleura, or abdominal viscera may be the starting point of the irritation, while in sensitive individuals, the action on the skin of a draught of cold air is sufficient to set up cough. Anæsthetic conditions of the larynx are occasionally met with in which such local irritations as those mentioned are not sufficient to excite cough; and depression of the activity of the reflex centre in the medulla, the result, for example, of the accumulation of carbonic acid in the blood, or of the action of opium, may diminish or completely abolish the act of coughing, and thereby cause a dangerous accumulation of secretion in the air-cells.

In examining cough as a symptom, it is well to note:

1st. Its Frequency and Rhythm.—Whether it comes frequently, each individual cough being separated by a tolerably constant interval, or whether there occur paroxysms of coughing with intervals of quietness.

2nd. Its Character.—This may vary very greatly. The cough may be dry, as in pleurisy, the first stage of phthisis, etc., or moist, as in chronic bronchitis, and in the last stages of phthisis. It may be painful, as in acute pleurisy, and it is important to observe that the patient then instinctively tries to suppress the cough which gives him so much pain; and this short, dry, suppressed cough is frequent at the commencement of acute pneumonia, and also in cases of intercostal neuralgia, pleurodynia, pericarditis, and peritonitis. Very different from this is the loud barking cough of hysteria, which is obviously produced at will, and calculated to attract the the utmost amount of attention. It is important to recognize the variety of cough met with in affections of the larynx. In laryngitis, even when the disease is very slight, the cough is hoarse, husky, stridulous, and croupy in character; and when much submucous infiltration has taken place, or if there be extensive formation of false membrane, the act of coughing is almost noiseless. The hard, metallic laryngeal cough met with in cases of aortic aneurism, when the recurrent laryngeal nerve is interfered with, is often of considerable diagnostic value.

3rd. Notice whether the cough is obviously brought on by such causes as exertion, change of posture, inhalation of cold air, of dust, or of irritating chemical vapors.

4th. Notice if the paroxysm terminates in a fit of vomiting, as often occurs in whooping-cough, phthisis, and chronic bronchitis, or in the prolonged, clear, shrill inspiration which characterizes the first of these affections.

Sputa.—A very important aid to diagnosis is found in the examination of the sputa, particularly at the commencement of morbid processes in the chest, when physical signs are not yet marked. In almost every affection of the respiratory organs, more or less expectoration follows the act of coughing. Occasionally, however, this is absent; and in the case of young children, even when the cough is accompanied with expectoration, the sputum is swallowed so soon as it reaches the mouth. It must also be borne in mind that the material coughed up does not always come originally from the respiratory tract; for secretions from the mouth, nose, and

pharynx may pass the rima glottidis, and, irritating the mucous membrane of the larynx, be coughed up again. Bleeding from the posterior nares may thus simulate hæmoptysis.

Chemical Characters.—As yet the chemical analysis of sputa has not proved of much diagnostic value. Consisting, in the main, of water, sputa have at different times been found to contain serum albumen, paraglobulin, paralbumen, myosin, nuclein, glycogen, various fatty acids, leucin, tyrosin, etc., in addition to the mucin which is invariably met with even in the healthy state, and which imparts to the expectoration its peculiar viscid and glassy appearance. Albumen is always present when there is inflammation of the air-passages or lung-substance. In cases of diabetes, sugar has been detected in the expectoration, and in renal affections urea may sometimes be found.

*Macroscopic Characters of Sputa.**

For purposes of ready description the various varieties of sputa may be classified as follows, each being named after its principal constituent.

(1.) *Mucous sputum* is transparent, clear and glassy, and has a viscid and ropy consistence which is best appreciated by pouring it from one vessel into another. It is sometimes present in health, often becoming constant in advanced life, but is most frequently found in the earlier stages of bronchial catarrh. There is a very slight admixture of mucous and pus corpuscles.

(2.) *Muco-purulent sputum* occurs in almost every affection of the bronchi and lung. When allowed to stand in a vessel, the pus corpuscles sink to the bottom, leaving the clear mucus floating on the surface. Sometimes, however, a more intimate mixture of these two elements takes place.

(3.) *Purulent sputum* possesses all the characters of ordinary pus as obtained from an abscess. It has the same yellow opaque appearance, and separates in the same way into two layers when allowed to stand, the lower being composed of pus corpuscles, the upper of plasma. This variety of sputum is usually derived from suppurating cavities in the

* In all cases of laryngeal and pulmonary disease the sputa should be regularly examined, and for this purpose the expectoration for twenty-four hours should be collected in a glass vessel of such shape as to permit of rapid and satisfactory inspection.

lung tissue, or is the result of other collections of pus (as, for example, empyæma) bursting into a bronchus.

(4.) *Serous Sputum* is that form which is met with when copious transudation takes place from the pulmonary circulation, as in œdema of the lungs. It has a characteristic thin, transparent appearance, and is usually copious and frothy.

(5.) *Sanguineous Sputum.*—The sputum may be simply streaked with blood (as in the early stages of phthisis, etc.), or the blood may be mixed intimately through the mass. This latter form is that most usually met with in the later stages of phthisis, in cases of hemorrhagic infarction, and in lobar pneumonia. In the last-named affection the sputum is of a rusty color, due to some chemical alteration of the blood pigment, and this may pass into citron-yellow and green. It has been already said that blood from the throat or posterior nares may trickle into the trachea and be coughed up. The primary source of the hemorrhage is then, however, usually clear. More difficult is it to distinguish hemorrhage from the lungs (hæmoptysis) from that from the stomach (hæmatemesis). The history and physical examination, and the nature of the act by which the blood reaches the mouth, will help greatly towards diagnosis; but it must be remembered that the blood coughed up may be swallowed and then vomited. In hæmoptysis the blood is usually bright red, fluid, and frothy, has an alkaline reaction, and when examined microscopically is found to contain more or less of those cellular elements which are peculiar to the respiratory tract. On the other hand, in hæmatemesis the blood is dark and venous; sometimes chocolate-brown, resembling coffee-grounds, often clotted, free from froth, acid in reaction, and when microscopically examined is found to contain fragments of food.

Physical Characters of the Sputa.

(1.) *Quantity.*—The amount of expectoration may vary very much, and this indication may become of considerable diagnostic value, as, for example, when in the course of some acute affection (bronchitis, pneumonia) the scanty sputum suddenly becomes more abundant and more readily expectorated, showing thereby that the acuteness of the inflammation is subsiding. In bronchiectasis very large quantities of sputum are brought up at one time, and so marked

is this symptom that it may suffice in many cases to establish a diagnosis in the absence of other signs.

(2.) *Form and Consistence.*—The more mucus the sputum contains, the firmer will be its consistence, and the more distinct its form. Tenacious sputa are consequently found in the acute stage of bronchitis, pneumonia, phthisis, etc. In the absence of mucus, the sputa lose their individual shapes, and, when collected in a vessel, they coalesce with each other. Such is the case with the purely purulent and the serous sputa. Tough sputa from phthisical cavities preserve their flattened, coin-like (nummular) shape after expectoration—an indication of some diagnostic value.

(3.) *Smell.*—As a rule, sputa are devoid of any very marked odor. When, however, putrefaction becomes developed, the odor of the breath and of the expectoration becomes most overpowering. This occurs to a marked degree in bronchiectasis, putrid bronchitis, and pulmonary gangrene.

(4.) *Color.*—To the yellow or yellow-green tinge which is imparted to the sputum by pus cells when they are present, allusion has already been made. The red color of sanguineous sputa has also been described, passing into rust-color, yellow, saffron, and finally green, as the hæmoglobin becomes more and more highly oxidized. Löwer has pointed out that in very hot weather a similar yellow tint is sometimes to be seen in the sputum, resulting from the development of a fungus (leptothrix). A yellow or a green discoloration frequently appears in the sputa in cases of jaundice, due to the presence of bile pigment; and those who are much exposed to smoke, or who work in coal-mines, frequently expectorate the black carbonaceous particles, which they have inhaled, to such an extent as to blacken the whole of the sputum.

Microscopic Examination of the Sputa.

(1.) *Pus, Blood, and Mucus Corpuscles.*—The recognition of these corpuscles is very readily made by means of the microscope. What diagnostic significance attaches to each has been already stated, and does not demand further remark.

(2.) *Epithelial Cells.**—The ordinary pavement epithelial

* These structures are best seen when stained with methyl-anilin. A small particle of the sputum should be mixed on the slide, with a drop of a watery solution (1-1000) of that pigment.

cells of the mouth are almost invariably present in sputa, becoming mixed with the expectoration on its passage through the mouth. They are of large size, polygonal in shape, finely granular, and possess a large, refractive, ovoid nucleus.

The columnar epithelium of the bronchial mucous membrane almost never appears in the sputa. When ciliated cells *are* present, they usually come from the nasal cavity.

Much more important for diagnosis is the occurrence of the epithelium of the pulmonary alveoli. As Friedländer* has shown, these alveolar epithelium cells never appear in the sputum in their normal flattened condition, but invariably swell up and become spherical when brought in contact with liquid, or when they undergo inflammatory change. In the sputa, this alveolar epithelium is readily recognized. The cells are spherical or slightly oval, have a diameter two to four times greater than that of a leucocyte (thus distinguished from all other round cells in the sputum), contain granular protoplasmic masses, and possess one or more large oval nuclei with distinct nucleoli. They further differ from all other cells to be found in the sputum in that they readily become pigmented, and undergo fatty and myelin† degeneration—changes which the other varieties seldom or never show. Bearing these points in view, the distinguishing of the alveolar epithelium can seldom be a matter of difficulty.

Regarding the diagnostic value of these cells,‡ it is important to observe that above the age of thirty to thirty-five years alveolar epithelium is to be found in the sputa of perfectly healthy persons, but that variety of cell is not found in individuals whose age is below thirty. At all ages, however, alveolar epithelium may be found in the sputa of many affections of the respiratory organs—in œdema, hypostatic congestion, hemorrhagic infarction, pneumonia, and in all the forms of phthisis. In simple bronchial catarrh of individuals under thirty, no alveolar epithelium

---

* *Ueber Lungenentzündung nebst Bemerkungen über das normale Lungenepithel.* Berlin, 1873.

† Panizza (*Deut. Arch.*, vol. xxviii., 1881, p. 343) argues strongly in favor of the view that this myelin transformation is mucous degeneration.

‡ On this subject see the very careful paper by Guttman and Smidt in the *Zeitschr. f. klin. med.*, iii., 1881.

is to be found in the sputa, unless the very finest bronchioles be affected, and then these cells appear only in small number. In commencing phthisical catarrh of the apex, however, alveolar epithelium is to be found in considerable quantity long before any physical sign can be detected, and in young individuals in whom all the other causes mentioned may be excluded, the occurrence of alveolar epithelium is almost certainly diagnostic of commencing phthisis.

(3.) *Débris of Lung Tissue.*—In any disease which involves destruction of lung tissue we may find in the sputum the elastic fibres which had formed the framework of the broken down alveolar walls. These fibres may be distinguished under the microscope without difficulty. They usually lie in groups coiled and twisted, sometimes recalling by their arrangement the outline of the alveoli. Their dichotomous branching and well-defined double contour, and still more, their resistance to the action of caustic alkalies, make their recognition a matter of little difficulty. It is well, as Fenwick recommends, to boil the sputa with caustic soda until the mixture becomes thin and watery. The elastic fibres will then readily sink to the bottom of a conical glass, and can be secured by means of a pipette.

While the débris of lung tissue occurs by far most frequently in the sputum of phthisis, it may also be found in cases of pulmonary abscess and of gangrene of the lung. In the last-named affection the lung tissue is only to be found in very fresh sputum. It rapidly disappears, being apparently acted upon and dissolved by some peculiar ferment which is present in the expectoration in such cases. It need hardly be said that where such elastic fibres occur, we have absolute proof of the destruction of lung tissue, hence the extreme importance of this symptom in case of phthisis where the physical signs are not distinct.

(4.) *Fibrinous Bronchial Casts.*—In pneumonia and in croupous bronchitis, there are to be found in the expectoration casts in fibrine of the finer bronchioles and their branches. In the sputum they are usually rolled together, and only unroll and spread out when washed with water. The perfect way in which they reproduce the arrangement of the bronchioles makes the recognition of these casts easy. In pneumonia they are most numerous in the sputum on the third and fourth day of the affection, and Remak, who devoted much attention to the subject, pointed out that the earlier these casts appeared, and the greater their quantity,

the more quickly would recovery set in and the more completely would the affected lung recover from the disease.

(5.) *Crystals* are occasionally met with in sputa, the most common being the long, fine, colorless, needle-shaped crystals of the fatty acids. They have some superficial resemblance to elastic fibres, but are easily distinguished by the fact that they dissolve at once in ether, a reagent which does not affect elastic fibre. These fatty acids are found in cases of putrid bronchitis, bronchiectasis, and pulmonary gangrene.

Another variety of crystal which may be found in the sputum are those usually known as *Charcot's crystals*, after the name of their discoverer. Their exact nature is a matter of some doubt. In shape they vary somewhat, but are usually long, fine, sharp, and spindle-shaped; they are colorless, are insoluble in alcohol, but are readily dissolved by acids or alkalies. Charcot's crystals occur most frequently in asthma, and have been supposed to be the exciting cause of the paroxysm, by irritating the terminations of the vagus.

Other crystals, such as cholesterin, hæmatoidin, leucin, tyrosin, oxalic acid, and triple phosphate, occur in the sputum, but do not demand special notice here.

(6.) *Micro-organisms* of various kinds may be found in the sputum, such as *leptothrix*, *oidium albicans*, and, rarely, *sarcina*. Bacteria and vibriones are very frequently to be seen in the sputum of gangrene and bronchiectasis.

By far the most important of these micro-organisms, however, is the *tubercle-bacillus*, which Koch has very recently* discovered. They are delicate, rod-shaped structures, in length usually about one-fourth or one-third the diameter of a blood corpuscle, and are motionless. They can only be detected after careful staining, and for this purpose the method of Ehrlich appears to be the most satisfactory. The procedure is as follows: A thin layer of sputum is spread on a cover-glass, which is then gently heated over a flame for a few seconds to coagulate the albumen, and placed in a staining solution prepared as follows: Five cubic centimetres of pure anilin are added to 100 cubic centimetres of distilled water, well shaken and filtered, and to the filtrate a saturated alcoholic solution of fuchsin or methyl-violet is added until precipitation com-

* *Berl. kl. Woch.*, 10th April, 1882.

mences. The cover-glass is allowed to float on this for half an hour. It is then washed in a solution of nitric-acid (1 to 2), and afterwards in distilled water. In this way the stain is extracted from everything but the bacilli. The detection of these tubercule-bacilli in the sputum suffices to establish a diagnosis of tuberculous affection of the lung.

Echinococcus-vesicles are in rare cases to be found in the expectoration, having either been previously encysted in the lung, or having bored their way from the liver into a bronchus.

(7.) *Foreign Bodies.*—To the presence of carbonaceous particles in the sputum allusion has already been made. Fragments of food, when accidentally present, are easily recognized by means of the microscope.

CHAPTER XIV.

Respiratory System—(*continued*).

EXAMINATION OF NARES AND LARYNX.

We now proceed to the physical examination of the organs of respiration, and these will be considered in the order in which they naturally come—the Nares, the Pharynx, the Larynx and Trachea, and the Lungs.

Nares.

The examination of the nostrils appertains more to the domain of surgery than to that of medicine; but it must be briefly alluded to here. Obstruction of the nasal passages obliges the patient to breathe through the mouth; and the effect of this upon the moisture of the tongue and lips has been already commented upon in Chap. I. The resonance of the nasal cavities is of great importance in connection with the voice; and when this resonance is interfered with by obstruction of the nares, the voice acquires a peculiar and characteristic nasal sound. The nares may be examined by—

(1.) *Palpation.*—The finger may to a certain extent be made to explore both anterior and posterior nares, and the

presence of polypi and other tumors may thus be ascertained.

(2.) *Inspection.*—The anterior nares may be inspected with the aid of a nasal speculum. With such an instrument it is often possible to examine the mucous membrane of the nostrils and the posterior wall of the pharynx, and even the orifices of the Eustachian tubes. The more posterior nasal structures are, however, best brought into view by means of *rhinoscopy*. This method of examination closely resembles laryngoscopy (to be presently described), the mirror is, however, turned in the reverse direction. It is passed over the back of the tongue until it touches the posterior wall of the pharynx, where it is held with the surface directed upwards and forwards. If the uvula hang loosely downwards it presents no impediment; but should it contract, turn backwards, and close up the posterior nares, it must be drawn forwards with a hook. Having in this way obtained a view of the posterior nares of the upper, middle, and lower passages, and of the openings of the Eustachian tubes, we must note the color of the mucous membrane covering these parts, and the presence or absence of swelling, ulceration, new formations, foreign bodies, etc.

Pharynx.

The pharyngeal passage, forming part of the alimentary tract as well as of the respiratory, has been already described in sufficient detail, and need not again detain us at this point.

Larynx.

Voice.—As an index of the state of the larynx, the voice is of the utmost importance. All affections of the vocal cords, whether ulceration, swelling, or new formation, and all acute and subacute inflammations of the larynx, are followed by huskiness of the voice. When therefore, the voice is clear and good, all such affections may be excluded. Aphonia (*a, priv., φωνή, sound*), or loss of voice, may, however, result from other causes, such as paralysis of the muscles of the larynx, which may be due to central or peripheral nerve affection, or simply to the exhaustion of severe disease; or it may be of a purely functional nature as met with in hysteria. A degree of aphonia may occur along with considerable dyspnœa owing to the embarrassment of

breathing; and where from central nervous disease the respiratory muscles are paralyzed, the voice fails.

Aphonia, must, of course, be distinguished from aphasia (loss of speech), in which the power of phonation is not affected, and also from deaf-mutism.

Laryngeal Palpation.—External palpation of the larynx may occasionally detect tender points, the result of inflammatory changes, which may be more clearly localized by laryngoscopic examination. By placing a finger on either side of the larynx whilst the patient speaks, the transmitted vibration of the vocal cords can be clearly felt. Normally this is equal on both sides, but should one cord be paralyzed, the vibration will fail on the corresponding side, and in this way a rapid and accurate diagnosis can sometimes be made.

Internal palpation of the larynx—that is, the examination of the rima glottidis and neighboring parts with the finger, is not so readily performed. The patient when seated in front of the operator must be made to bend the head back, to open wide the mouth, and to thrust out the tongue. The right forefinger of the operator must then be passed rapidly backward along the roof of the mouth, and then suddenly bent downwards until its tip comes in contact with the epiglottis and neighboring parts. This method of examination is chiefly useful to detect the presence of œdema glottidis, and of tumors lying over the orifice of the larynx, and to remove foreign bodies which may have become lodged there.

Laryngoscopic Examination.—It is not necessary here to describe the various forms of laryngoscopes which have been devised, and many of which are now in use by different observers. For general purposes it suffices to have three or four throat mirrors of different sizes, which are plane mirrors, usually round in shape, attached at an angle to their metallic rods, which fit into an ivory or wooden handle, and are there secured by means of a screw. It is of some importance to reserve one of these mirrors for syphilitic cases. The light used for illumination may be sunlight or daylight, but in this climate it is better to trust to the artificial light of a good lamp. The rays of light are reflected into the patient's mouth from a concave mirror, which is most usually secured to the forehead of the operator by means of a circular band passing round the head. This mirror has a small opening in the centre, which is placed so as to correspond to the

eye of the observer, and through which he can see the laryngeal mirror.

Method of Examination.—The patient must be seated opposite and very close to the observer, and the fauces illuminated from the mirror on the forehead of the latter. The light should be placed near the side of the patient's head, at the level of and slightly behind the mouth. The patient having been made to open his mouth widely, and to protrude his tongue, grasping its point between the thumb and forefinger of his right hand, protected with a handkerchief or napkin, the observer now takes up the laryngeal mirror, which has been previously warmed, in the right hand (he ought to be able to use the left hand also for this purpose), and, holding it as one holds a pen, he passes it rapidly into the patient's mouth, and, carefully avoiding unnecessary contact with the surface of the tongue or palate, presses its posterior surface upon the uvula. It will be found to increase the steadiness of this movement if the little finger of the operator be allowed to rest on the patient's cheek at the angle of the mouth. With the mirror lying in this position the larynx usually comes more or less perfectly into view, when the patient is desired to ejaculate "ah." By slight movements of the mirror the whole of the larynx can be explored. Turning the laryngeal mirror downwards, there come into view in succession the back of the tongue, the upper surface of the epiglottis with the glosso-epiglottidean ligaments, the arytenoid cartilages, the false and the true vocal cords, the ventricles of the larynx, the rings of the trachea, sometimes even as far down as the bifurcation. There are several difficulties which may arise in the course of laryngoscopic examination. Apart from the obstruction occasioned by enlarged tonsils, the observer may have to combat incessant attempts to retch, caused by the contact of the laryngeal mirror with the throat, especially when that region is sensitive. This can only be overcome by patiently accustoming the parts to the abnormal irritation by means of repeated examination. In other cases the back part of the tongue may arch upwards to such an extent as to shut out the view. Very frequently this difficulty disappears when the patient is directed to say "ah," but if this measure fails, then the organ may be forcibly depressed by means of a spatula.

It is to be borne in mind that the parts reflected in the right and left part of the mirror correspond to the *patient's*

left and right, and that the more anterior structures are seen in the upper part of the mirror, the more posterior in the lower.

Having carefully examined the various portions of the larynx, which may be brought into view by changing the position of the mirror, it is perhaps best to systematize the information gained by grouping the facts under some such heads as the following :

(1.) *Changes in Color.*—In the normal larynx the mucous membrane generally has a clear red appearance, while the epiglottis is slightly yellow, and the true vocal cords stand out distinctly, having a clear pearly-white color. In anæmic conditions, the interior of the larynx generally becomes paler in color; whereas in acute catarrhal conditions the parts assume an intense red, especially the epiglottis, and even the true cords lose their whiteness and become swollen, red, and injected. In more chronic catarrh the tint assumed is not so bright, but is more grayish red. In cases of croup the false membrane can sometimes be seen in the larynx.

(2.) *Ulceration.*—If ulcers are visible, their position, size, and general features should be noted.

(3.) *Tumefaction.*—Swelling of the parts round the glottis (*œdema glottidis*) may occur from a variety of causes, and the early recognition of this very dangerous condition by means of the laryngoscope is of the utmost importance. New formations are sometimes met with, and their size, character, and seat must be carefully noted.

(4.) *Foreign bodies.*—In children, in particular, the laryngoscope is of great service in showing where a foreign body has lodged, and in aiding in its removal.

(5.) *Movements of the Larynx, particularly of the True Cords.*—The larynx is supplied by means of two nerves, the superior and the inferior (or recurrent laryngeal branches of the vagus). Paralysis of these nerves produces different symptoms, and must be considered separately. The movements of the cords are very readily discerned. During inspiration they move apart, so as to leave a very free passage for the air, while during expiration near each other again. These movements are, of course, exaggerated during forced breathing. When a note is sung the vocal cords of each side approach very close to each other, so as almost to come in contact.

Paralysis of the Superior Laryngeal Nerve causes anæsthesia of the mucous membrane of the upper and middle portions of the laryngeal cavity, along with paralysis of the crico-thyroid muscle, and of the depressors of the epiglottis (thyro and aryteno-epiglottidean muscles). As a consequence of this the epiglottis is immovable, and applied to back of the tongue; and as without the action of the crico-thyroid the vocal cords cannot be rendered tense, the voice is hoarse and deep, and (according to Mackenzie) the cords can be seen to be loose, their centre being visibly depressed during inspiration, and elevated during expiration. The paralysis of the epiglottis allows particles of food to enter the larynx. Paralysis of the superior laryngeal nerve is usually the result of diphtheria.

Paralysis of the Inferior (or Recurrent) Laryngeal Nerve may be due to central degenerative changes in the floor of the fourth ventricle (in bulbar paralysis, and other affections of the pons and medulla), but it is much more frequently the result of lesions of the nerve trunks in their course. Whilst the two recurrent nerves are equally liable to suffer from the pressure of such tumors as bronchocele, cancer of the upper part of the œsophagus, etc., it is to be carefully borne in mind that the course of the left nerve exposes it specially to injury from the pressure of aortic aneurisms, while the right recurrent is frequently paralyzed by being involved in thickening of the right pleura with which it lies in contact—a condition met with in phthisis of the right apex.

Bilateral Recurrent Paralysis.—In this very rare condition the vocal cords are perfectly immobile, and may be seen to have assumed what Von Ziemssen calls the cadaveric position, that, namely, which is found in the dead subject;* there is absolute loss of voice, and the patient speaks in a whisper, and that with considerable exertion and difficulty, owing to the great expenditure of air required on account of the width of the glottis. For the same reason coughing becomes extremely difficult, but there is little or no dyspnœa.

Unilateral Recurrent Paralysis.—In this condition the vocal cord on the paralyzed side occupies the cadaveric position already described, while the healthy cord possesses

* The cords lie about midway between the lateral position of deep inspiration and the median one of phonation.

its normal range of movement, and, indeed, rather exceeds this, crossing the median line to some extent so as to compensate for its paralyzed neighbor. The voice is impure, metallic, and high pitched, and readily, as Traube pointed out, passes into a falsetto.

Paralysis of the Individual Muscles supplied by the Inferior Pharyngeal Nerve.

(1.) *Posterior Crico-Arytenoid Muscles.*—These muscles have for their function the widening of the glottis, which is necessary for inspiration. When they are both paralyzed a condition ensues which is one of the gravest met with in connection with laryngeal pathology. The two vocal cords are then found to be lying close to each other in the middle line, and from this position they do not move even during inspiration. The consequence is, that when the paralysis is complete there is very well marked inspiratory dyspnœa, and this not merely because the vocal cords cannot be drawn asunder by the paralyzed muscles, but because the rush of air forces them still more closely together. While inspiration is, therefore, noisy, the voice is usually unaffected.

When only one of the posterior crico-arytenoid muscles is paralyzed, the vocal cord of the affected side lies in the middle line; the voice is impure, but the glottis being comparatively wide, it is only with forced inspiration that there is any noise.

(2.) *Arytenoid Muscles.*—These muscles having for their function the closure of the posterior third of the glottis, it will be easily understood that when they are paralyzed both vocal cords lie during phonation in their normal position for the anterior two thirds of their length, while at the posterior end of the glottis an open triangle is left through which air escapes unhindered. As a consequence of this the voice is hoarse and impure.

(3.) *Internal Thyro-Arytenoid Muscles.*—The action of these muscles is to render the vocal cords tense, and thereby to close the glottis. When one is paralyzed, the cord of the corresponding side is lax, and shows a slight concave excavation of its inner edge. When the paralysis is bilateral, this excavation is of course found in both cords; the voice becomes hoarse, and speaking difficult.

The symptoms of individual paralysis of other muscles of the larynx have not yet been clearly ascertained.

CHAPTER XV.

Respiratory System—(*continued*).

INSPECTION.

In order to determine the position of any particular point on the thoracic wall for the purpose of description or record, the thorax has been divided arbitrarily into certain regions, which may be grouped in the following manner:

1. *Median or sternal group*, bounded on either side by the sternal border, which comprises—

(*a.*) Supra-sternal notch.
(*b.*) Superior sternal region.
(*c.*) Inferior sternal region. The two last regions are separated by a horizontal line corresponding to the level of the lower border of the third costal cartilage.

2. *Antero-lateral group*, bounded internally by the sternal border, and externally by a line which commences at the first ring of the trachea, runs diagonally outwards to the acromion process, and then falls vertically downwards. This group comprises—

(*a.*) Supra-clavicular region lying above the upper edge of the clavicle.
(*b.*) Clavicular region, corresponding to the inner half of the clavicle.
(*c.*) Infra-clavicular region, from the clavicle to the lower border of the third rib.
(*d.*) Mammary region, from the third to the sixth rib.
(*e.*) Infra-mammary region, from the sixth rib downwards.

3. *The lateral group* corresponds to the axilla, being bounded anteriorly by the vertical acromial line, which limits the antero-lateral group, and posteriorly by the axillary border of scapula. This group comprises—

(*a.*) Axillary region.
(*b.*) Infra-axillary region, which is separated from the former by a horizontal line at the level of the sixth rib.

4. *Posterior group*, bounded externally by the axillary border of the scapula, and internally by the middle line posteriorly. The members of this group are—

(*a.*) Supra-scapular region, lying above the scapula.
(*b.*) Supra-spinous region, corresponding to the supra-spinous fossa.
(*c.*) Infra-spinous region, corresponding to the infra-spinous fossa.
(*d.*) Infra-scapular region lying below the scapula.
(*e.*) Inter-scapular region, lying between the scapula and the middle line.

Inspection.

For the inspection of the thorax the patient should be placed in a good light, if possible in a sitting posture in an unconstrained position, and with the surface of the chest fully exposed. The general outline of the thorax ought to be viewed from the front, from the back, from either side, and from above and behind, looking downwards. Such inspection gives information concerning (1) the form, and (2) the movements of the chest.

1. **The Form of the Chest.**—The typical chest formation, which is, however, but rarely met with, may be said to possess the following characteristics. Conical in form, with the antero-posterior diameter shorter than the transverse, it is symmetrical on both sides, both generally, and at each corresponding point. The supra- and infra-clavicular regions are almost on a level with the clavicles, and from the collar-bones downwards to the fourth rib there is on either side a gentle convexity. The nipple is placed (in the male and virgin female) on the fourth rib or fourth intercostal space, and from this point downwards the chest wall becomes somewhat flattened. In the upper two thirds of the chest the outlines of the ribs are not well defined, but below this the thinner covering of muscle allows their form to become apparent. The spine and sternum occupy an almost exactly median position, and the shoulder-blades are symmetrical.

From this typical form there are many deviations compatible with health (physiological heteromorphisms, as Woillez * terms them), of which the principal are those associated with the phthisical and with the rickety constitutions. Many persons who are predisposed to phthisis show a peculiar thoracic conformation which has been called alar, or pterygoid, on account of the wing-like projection of the scapulæ. The chest is long, narrow, sometimes flattened anteriorly, the ribs oblique, the shoulders sloping, and the throat prominent.

The occurrence of any obstruction to the respiration in childhood along with rickets tends to produce the "pigeon breast," through the yielding of the softened ribs. In this form of thorax the ribs are straightened, and the sternum thrown forwards so that a transverse section of the chest would approach a triangular form. Independently, however, of any pulmonary complication, rickets may of itself determine a peculiar thoracic formation, when the ribs are so soft as not to be capable of bearing the atmospheric pressure necessarily thrown upon them during inspiration. A longitudinal groove is thus formed on either side of the sternum.

Irregular formation of the thorax may also be caused by deformities of the spinal column.

Changes in the Form of the Thorax in Pulmonary Diseases.

These may be local or general.

(1.) *Local.*—Bulging is met with in encapsuled pleural effusions, in empyæma, in pericardial effusions, in cardiac hypertrophy, and over large cavities in the lung. Tumors of the liver and spleen may also cause bulging, the former at the right side, the latter at the left, and surgical affections of the chest wall may give rise to local swelling. Localized shrinking occurs chiefly in connection with phthisis, when there may be flattening in the supra- and infra-clavicular regions. The rare condition in which there is congenital absence of part of the pectoral muscles must not be mistaken for flattening.

(2.) *General.*—Bilateral enlargement of the thorax results from pulmonary emphysema. This so-called barrel-shaped chest is enlarged in all its diameters, rounded, and the in-

* *Traité de Percussion*, etc., p. 415. Paris, 1879.

tercostal spaces wide. The respiratory movements are very slight, and the thorax remains permanently in a condition resembling that of full inspiration. Unilateral enlargement may arise from extensive pneumonia, from tumor affecting the greater part of one lung (Eichhorst), but it is most evident when effusion of fluid or gas takes place into the pleural cavity. In pleurisy with extensive effusion the diameter of the thorax on the affected side is increased; the intercostal spaces are wide, and rise to the level of the ribs, or even bulge beyond them; the nipple is moved upwards and outwards, and the heart is pressed over towards the sound side in the manner already described. Unilateral shrinking of the chest may come on as the result of absorption of a pleural effusion when the lung is not in a condition to expand. It is also met with in cases of pulmonary cirrhosis.

2. **Respiratory Movements.**—In connection with the act of breathing we have to note the following points—(1) its frequency, (2) its rhythm, (3) its type, (4) its pain or difficulty, (5) the extent of the movements.

(1). *The Frequency of Respiration.*—The respiratory movements are so much under the control of the will that the physician should endeavor to estimate their rapidity without the knowledge of the patient. This is best done by holding the fingers upon the radial artery, as if to count its pulsations, while the patient's hand rests upon the epigastrium and rises and falls with the respiration. Whilst in new-born children the breathing may be at the rate of forty-four per minute, in the adult male it averages from sixteen to twenty-four, but is slightly more rapid in the female. It is increased in rapidity by exertion, and after meals, and is slower in the recumbent posture than when sitting or standing. It reaches its lowest rate during sleep. It is most important to note the ratio between respiration and pulse which is usually 1: 4, but may vary from 1: 1 to 1: 7.

Pathologically, the act of breathing is rendered slow by stenosis of the larynx (as in croup), and by any cerebral disease which induces pressure upon the respiratory centre in the medulla. More common, however, is increase in frequency, which may arise in a variety of ways—(1) When the act of respiration causes pain (as in pleurisy and peritonitis); (2) reflexly, as a result of pain situated in other organs; (3) from chemical changes in the blood, such as are met

with in anæmia and in lucocythæmia; (4) from mechanical hindrance to the entrance of air into the pulmonary air-cells, which may exist in the larynx, trachea, bronchi, or lung tissue; or may result from pressure on the lung from without by means of pleural effusion, ascites, meteorism, etc.; (5) disturbance of the circulation through the lungs, which may arise from a variety of conditions, such as valvular disease, pulmonary embolism, etc.; (6) from fever (the increased temperature of the blood over-exciting the respiratory centre—the so-called "heat-dyspnœa"); (7) from certain nervous disorders, such as hysteria.

(2.) *The Rhythm of the Respiratory Movements.*—In health the rhythm of the breathing, when uninfluenced by will, is very regular, expiration following inspiration immediately, and being somewhat shorter in duration, after which there is short a pause. Walshe* calculates that if the whole act be taken as equal to 10, then the inspiration may be estimated as 5, the expiration as 4, and the pause as 1. These relations, however, only hold good in health. In disease either the expiration or the inspiration may be altered in duration usually at the expense of the pause. Inspiration is lengthened whenever an obstacle to the entrance of air exists in the larynx or trachea, and is particularly well marked in cases of paralysis of the posterior crico-arytenoid muscles. Expiration on the other hand is prolonged when any obstruction to the exit of air exists in any part of the respiratory tract, as is the case in vesicular emphysema. The rhythm of the respiratory movements frequently becomes jerking and unequal, particularly in children where the flexible chest wall yields to the external atmospheric pressure during inspiration, when any obstruction to the free entrance of air exists in the larynx, trachea, or bronchi.

One of the most peculiar alterations in rhythm is seen in the Cheyne-Stokes breathing, when the sequence of the recurring respiratory acts is broken by the occurrence, at intervals of about 1 to 1½ minute, of pauses, during which respiration entirely ceases. These pauses, which last from ¼ to ¾ of a minute, are followed by the gradual resumption of the respirations, which, at first short and superficial, grow gradually deeper up to the point of dyspnœa, after which the breathing becomes again shallower until the next pause is reached, and so on. The exact manner in which this

* "Diseases of the Lungs," 4th edition, p. 14.

peculiar rhythm is produced is somewhat doubtful. One most important factor seems to be a deficient supply of oxygen to the respiratory centre in the medulla. The Cheyne-Stokes breathing is met with in many cerebral diseases, in uræmia, in fatty degeneration, in valvular disease of the heart, and is usually one of the immediate precursors of a fatal termination.

(3.) *The Type of the Respiratory Movements.*—In men the respiratory movements chiefly affect the abdominal walls and the lower ribs (costo-abdominal type), while in women the diaphragm does not take so prominent a part in the act of breathing, and the movement is in great measure confined to the upper part of the thorax (costal type). In disease, however, these conditions may be changed, for anything which interferes with the movements of the diaphragm (such as ascites, peritonitis, and many other affections of the abdomen) will in a man change the type of breathing into the purely costal, whilst the latter type of breathing may be lost in a woman when there is some painful affection of the thoracic organs which obliges the respiration to be chiefly abdominal.

(4.) *Pain and Difficulty in Breathing.*—Pain in relation to the organs of respiration has already been mentioned, and need not detain us here. When present, it is usually, though not always, aggravated by the respiratory movements.

Dyspnœa, or difficulty of breathing, may be of a purely nervous or subjective kind. The rate is then usually normal, and the air can be heard to be freely entering the lung, while all the causes of objective dyspnœa are absent. This symptom usually occurs in hysterical women. In true objective dyspnœa the respirations are deep and long drawn, and, as already mentioned, either the inspiration or the expiration may be interfered with. In the former case, all the accessory muscles of inspiration are called into play, and their contractions form a very striking feature in such cases. These include not merely the dilators of the thorax, such as the sterno-mastoids, the scaleni, the pectorals, etc., but also those muscles which dilate the nostrils, elevate the soft palate, depress the larynx, and open the glottis. In order to the effective action of these accessory muscles, the patient has to assume the sitting posture (orthopnœa), and, provided that he is not comatose, the degree of difficulty of breathing which is present may be more or less accurately

estimated, according as the position assumed approaches the sitting posture.

(5). *The Extent of the Movements.*—In the barrel-shaped chest which accompanies vesicular emphysema, there is, as has been already said, diminution of the movements of the chest in all directions. More important, however, for the purposes of diagnosis, are those localized inequalities in the range of the movements which are occasionally met with. When one lung is compressed by reason of pleuritic effusion, or is from any other cause rendered incapable of expansion, the thoracic movements on that side become defective. Phthisical consolidation at the apices gives rise to deficient movement in the upper part of the chest, as compared with the lower; while in cases of stenosis of the larynx and in emphysema, the opposite condition obtains, for then during the expansion of the chest there is depression of the lower intercostal spaces, of the epigastrium, in the supra-clavicular regions, and in the supra-sternal notch.

CHAPTER XVI.

RESPIRATORY SYSTEM.—(*continued*).

PALPATION.

BY laying the hand flat upon the thorax and palpating its walls, information may be obtained regarding the form and movements of the chest, the presence or absence of fremitus, of fluctuation, and of certain pulsatory movements other than those already referred to in connection with the circulatory system.

1. **The Form of the Thorax.**—The general form of the chest is best appreciated by means of simple inspection; but localized changes in shape may be recognized by palpation.

2. **The Movements of the Thorax.**—The information obtained by inspection may be supplemented by laying the

hands on the thorax, and estimating the local movements of expansion and elevation at particular parts.

3. **Vocal Fremitus,** or that vibration of the chest wall which may be felt in a healthy person while speaking, is of considerable diagnostic importance. Under the vocal cords in the larynx lies an air column, which extends through the trachea and bronchi to the pulmonary alveoli, and which is set in vibration when the vocal cords vibrate, and through the bronchial walls and lung tissue the thrill so generated is conducted to the thoracic parieties. It is not difficult to understand the conditions under which its intensity becomes increased or diminished. The thickness of the thoracic wall has an important influence, the thrill being more distinct in emaciated subjects than in those who have much deposit of fat underneath the skin. The intensity of the vocal fremitus also depends upon the loudness of the tone spoken, and upon the depth of its pitch; and finally, it must not be forgotten that it is more distinct in men than in women and children, and that the thrill on the right side is almost invariably greater than that on the left, this being accounted for by the larger calibre of the right bronchus.

In disease the vocal fremitus may be diminished or increased.

(1.) *Diminished.*—Any condition which blocks up the bronchi, such as collection of mucus or pus, or compression by means of tumors, will produce a diminution of the vocal fremitus over the corresponding part of the chest wall. More extensive loss of the thrill is met with where effusion of fluid or gas into the pleural cavity has taken place. If the effusion be extensive, the vocal fremitus may be entirely lost; and should the lung be bound down by extensive adhesions, the fremitus may not be regained even after the entire absorption of the effusion.

(2.) *Increased.*—When infiltration takes place into the air-cells of the lung, the pulmonary parenchyma becomes at once a better conductor of the vocal vibrations, and in consequence the thrill becomes intensified. Such is the case, for example, in lobar pneumonia; and where the lower lobe is affected, the vocal fremitus gives most important aid in distinguishing that affection from pleural effusion. The fremitus is likewise increased where there is phthisical consolidation, particularly if cavities have formed; but should

a main bronchial branch leading to the part have become obstructed, either by pressure or by the collection of mucus, pus, or blood, the vocal thrill may be diminished or lost.

4. **Pleural, Bronchial, and Cavernous Thrills.**—The palpating hand may also detect the fremitus occasioned by the rubbing together of the roughened pleural surfaces in cases of pleurisy, and large râles in the bronchi or in cavities in the lungs may communicate an appreciable thrill to the walls of the chest. These are, however, of little diagnostic importance, in that they are better appreciated by the aid of auscultation.

5. **Fluctuation.**—When one side of the thorax is distended with fluid, fluctuation may occasionally be detected in it, more particularly and importantly in empyæma.

CHAPTER XVII.

RESPIRATORY SYSTEM—(*continued*).

MENSURATION.

MENSURATION, which is intended to render precise the information which may be gained by inspection and palpation, and which in some of its developments passes much beyond these, is performed by the aid of a variety of instruments which fail to be described in detail.

1. **The Tape-Measure** is used to ascertain the circumference of the chest, which, at the level of the nipples and at the end of expiration, measures in the healthy male adult about thirty-two or thirty-three inches. A full inspiration increases this figure by from two to five inches, while in quiet respiration the inspiration position exceeds the expiration by about half an inch. Unfortunately the circumferential measurement of the chest is of comparatively little diagnostic value, as very great variations are met with in health. Much more important is it to learn the relative size of the two sides of the chest. This is most conven-

iently done by joining two tapes at the commencement of their scales, and fixing this point of junction over the vertebral column. Each side of the chest has thus a tape for itself, and the two measurements can be simultaneously made and compared. In right-handed persons the right half of the chest is about half an inch larger in circumference than the left; while in those who are left-handed these conditions are either reversed, or, what is more common, the two sides of the chest are practically identical in size. Unilateral enlargement and shrinking, the result of disease, are very readily detected by means of such measurement.

2. **Callipers.**—Various diameters of the chest may be measured by means of a pair of common steel callipers. Of these the most important is undoubtedly the antero-posterior (sterno-vertebral), which in the phthinoid chest is much diminished—the normal measurement being 9 to 10 inches. More difficult is it to obtain exact measurements of the antero-posterior diameter of either apex. For this purpose, one point of the callipers is to be applied immediately below the centre of the clavicle, and the other on the spine of the scapula at a similar distance from the middle line. If great care be taken, sufficiently reliable results may in this way be obtained, when it will be found almost invariably, that in healthy persons the right measurement very slightly exceeds the left. An excess of even a fourth of an inch on the right side indicates, however, morbid depression on the left, while, if the left be in excess by that amount, there is still more conclusive evidence of contraction on the right side.*

3. **Cyrtometer.**—This instrument, devised by Woillez, consists of a series of small pieces of whalebone, so articulated together as to form a stiff chain which, when closely applied to the walls of the thorax, retains the curves given to it, and which, when removed and laid upon a large sheet of paper, permits of these curves being marked out on the paper. The instrument may be more simply constructed of two pieces of lead wire, joined together by means of a piece of india-rubber tubing, and in this form it is very

* Walshe, "Diseases of the Lungs," 4th ed., p. 33.

easily used, and the outline of the chest wall can be most accurately depicted by its means.

So far I have described those instruments which are fitted to give us measurements of the chest when at rest, and I now proceed to consider those which have for their design the measurement or registration of the thoracic movements.

4. **Thoracometer, or Chest-Measurer.**—This instrument, as constructed by Sibson,* consists, in its essential parts, of a dial which measures accurately the vertical movements of a small rod, which is applied to the surface of the chest by means of a spring. Owing to various errors which are necessarily present in the readings obtained, this instrument has never come into general use.

5. **Stethograph.**—The double stethograph of Riegel appears to be more trustworthy in its results than the last-named instrument. Two levers, which are acted upon by the movements of the chest walls at two different points, are so arranged as to record their results on a strip of paper, travelling horizontally by clockwork. The tracings so obtained enable us to analyze the respiratory movements in a much more exact manner than can be done by means of any other instrument. Pathologically, the most striking changes are those in which there is an impediment to the free entrance or exit of air. For example, where the larynx or trachea is stenosed, while the expiratory curve is normal, the inspiratory is much prolonged. The reverse is the case in emphysema, where the expiratory curve is prolonged and irregular.

I now come to the third class of instruments of mensuration, those, namely, which deal with the air passing into and out of the chest.

6. **Spirometer.**—Hutchinson's spirometer consists of a gasometer properly poised and adjusted, into which the patient expires forcibly through an elastic tube, and which is arranged so as to measure the amount of expired air. The "vital capacity" varies with age, stature, and sex, but when allowances have been made, it may be said, as a general rule, that a diminished quantity of air is expired when there is stenosis of larynx, trachea, or bronchi, inter-

* *Medico-Chirurgical Transactions*, 1858.

ference with the free movement of the thoracic walls, or diminution of the respiratory surface of the lungs. Of these diseases the most striking in its results is undoubtedly phthisis.

Hutchinson gives the following table of the results he obtained from very numerous observations:

Stature.	Capacity of Healthy Males.	Early Stage of Consumption.	Advanced Stage of Consumption.
	Cubic Inches.	Cubic Inches.	Cubic Inches.
From 5 feet to 5 feet 1 inch,	174	117	82
" 5 " 1 inch to 5 " 2 "	182	122	86
" 5 " 2 " to 5 " 3 "	190	127	89
" 5 " 3 " to 5 " 4 "	198	133	93
" 5 " 4 " to 5 " 5 "	206	138	97
" 5 " 5 " to 5 " 6 "	214	143	100
" 5 " 6 " to 5 " 7 "	222	149	104
" 5 " 7 " to 5 " 8 "	230	154	108
" 5 " 8 " to 5 " 9 "	238	159	112
" 5 " 9 " to 5 " 10 "	246	165	116
" 5 " 10 " to 5 " 11 "	254	170	119
" 5 " 11 " to 6 "	262	176	123

It must, however, be borne in mind that there are many fallacies in the use of this instrument. Some persons cannot be made to understand how to blow, others by taking great pains attain to higher figures than their average, and finally, by practice, the art of blowing is so readily learned that those accustomed to the instrument can raise the gasometer cylinder to very considerable elevations.

7. **Pneumatometer.**—This instrument, by means of which the force of expiration and inspiration is measured, is most conveniently used in the form devised by Waldenburg, which consists of a simple mercurial manometer connected by means of an elastic tube with a mouthpiece which fits accurately and tightly over the mouth and nose of the patient. When the patient expires through the tube, the column of mercury sinks in the proximal limb of the manometer and rises in the distal, while with inspiration these movements are of course reversed, and in either case the amount of displacement is to be read off on the scale.*

* Since the level of the mercury when at rest corresponds in both limbs to the zero of the scale, the reading obtained must, of course, be doubled to represent the true difference in the level of the two columns.

In a moderately well-developed male the inspiratory pressure is from 70 to 100 mm. of mercury, while the expiratory is from 90 to 130 mm., whereas in the female we get with inspiration a pressure of 50 to 80 mm. and with expiration 70 to 110 mm. Of more importance than the absolute pressure (which varies much in different individuals) is the difference between the expiratory and the inspiratory pressure, and it must be carefully borne in mind by those who use this very valuable instrument that in healthy persons the power of expiration exceeds that of inspiration by 20 to 30 mm. It is by comparing these two pressures that the most important indications are obtainable. Their relation is altered in disease as follows:

Expiratory pressure is increased in relation to inspiratory in phthisis (even at a very early stage), in stenosis of the air passages, in pulmonary congestion, pneumonia, and pleurisy, and in such abdominal affections as impede respiration by pressing the diaphragm upwards.

Expiratory pressure is diminished until it becomes equal to or below the inspiratory in pulmonary emphysema.

Undoubtedly, the importance of the diagnostic indications given by the pneumatometer in cases of incipient phthisis is very considerable, and in all chronic chest complaints the instrument is of value in indicating the progress of the disease—progressive or retrogressive, as the case may be—and in estimating the results of treatment.

The space at my disposal does not permit of a description of various other, less important, instruments.

CHAPTER XVII.

RESPIRATORY SYSTEM—(*continued*).

THEORY OF PERCUSSION.

WHEN the surface of the chest is percussed in the manner which will be described in the next chapter, a sound is produced which is called the percussion note of the part. This term *note* is, however, apt to mislead, for it is not a simple or pure note, not being composed of a regular series of simple vibrations, nor is it (as is the case with the sounds

produced by musical instruments) made up of one well-marked basal or fundamental tone and a series of higher-pitched upper partial tones which bear a definite relation to the basal or prime tone. The sound which is heard on percussion of the healthy chest is composed of a large number of tones, bearing no definite relation to one another, and in it no definite or well-marked fundamental note can be distinguished. It is very often assumed that the note produced on percussing the front of the chest—for example, at the level of the second rib—is made up of a distinguishable fundamental or prime tone, corresponding in pitch to the antero-posterior diameter at the particular point in question, just as when one blows across the mouth of a test-tube the prime tone obtained corresponds in pitch to the length of the air column contained in the tube. This view is, however, clearly untenable; for, apart from the inherent improbability of this particular air column alone being set in audible vibration, and not the many others which radiate from the point of percussion to the other limits of the thoracic cavity, there is the fact that in practice it is impossible to find what is the real pitch of this fundamental or prime tone; and, further, it is constantly noticed that the apparent pitch of the percussion note varies enormously with the variety of plexiimeter employed, and still more when the plexiimeter and finger are compared, which would not be the case were there a distinguishable prime tone.

The percussion note is made up of vibrations which are derived from three sources.

1. *The Vibrations of the Plexiimeter.*—When the finger is employed as a plexiimeter, these vibrations are practically inaudible. In the case of an ivory plexiimeter, however, they are readily recognized. If the instrument be of the usual form, the vibrations are clear and relatively high in pitch; but provided that the plexiimeter be properly damped by being firmly pressed upon the thoracic wall, and be struck with the pulp of the finger alone, or with the india-rubber of a hammer, the tone it gives can be readily discounted by the physician.

2. *The Vibrations of the Thoracic Wall.*—These are of so ill-marked a character (unless the point struck lie over the rib of a very thin subject) and have so little intensity as compared with the intrathoracic note, that in themselves they need hardly be considered, though, as I will presently point out, the condition of the chest wall and its vibrations when

percussed have a very important influence on the character of the intrathoracic note.

3. *The Vibrations of the Air in the Lungs.*—These vibrations constitute the important part of the percussion note, and must be considered in some detail.

When percussion is made at any point of the chest wall, the air in the lungs is set in vibration, and the point which is struck may be considered as the point of divergence of a series of radiating air columns whose lengths may be represented by lines drawn from the corresponding point on the visceral pleura to the opposite walls of the thorax in all directions. The lengths of these very numerous columns, of course, differ considerably; and since an air column, when set in vibration, produces a note proportionate in pitch to the length of the column, the numerous notes which go to make up the percussion sound vary considerably in pitch. The pulmonic septa also, in all probability, limit the length of certain of these air columns, and in others they may determine nodal points, and in this way cause still greater differences in pitch. We have thus to consider the intrathoracic percussion sound as composed of a large number of prime or fundamental tones, which vary much in pitch, and each have an ascending series of upper partial tones which tend, of course, still further to render the vibrations of the combined percussion note irregular.

In a musical note we have to recognize three distinct characters—viz., intensity, pitch, and quality; and in relation to the percussion sound, these must also be considered.

1. *Intensity.*—The intensity of a musical tone depends upon the amplitude of the individual vibrations of which the tone is composed. In the case of the drum, for example, the intensity of the tone depends upon the vigor with which the drum-head is struck. In the same way, the intensity of the percussion-note depends, to a considerable extent, upon the strength of the stroke. But it must be remembered that the percussion note, as I have just said, is composed of a large number of different tones, so that its intensity in any given case depends also upon the number of these tones which are produced by the blow. Thus, when the greater part of a lung is hepatized, the percussion note over the healthy portion loses much of its intensity, because there are fewer air columns which can be set in vibration.

The intensity of the note is, therefore, of considerable diagnostic significance.

2. *Pitch.*—The pitch of a simple tone, such as that of a tuning-fork, depends upon the rate of the vibrations of which it is composed. In the case of a musical note, composed of a prime tone and an ascending series of upper partial tones (as, for example, the note of a stretched cord), the term pitch is understood to mean the pitch of the prime tone. It is thus clear that, in regard to the percussion sound, we cannot, properly speaking, use the term pitch, since it is impossible to detect any fundamental or prime note. It is evidently advisable, however, to retain the term, which is so useful clinically, provided that in using it we carefully keep in mind that we do not refer to the pitch of a basal note, which, as I have said, does not exist, but to the general pitch of the combination of tones which reach the ear. If we take an illustration from the piano, it will easily be seen what is here meant. Suppose that a number of notes at the treble end of the key-board be struck simultaneously with one or two at the base end, the general impression will be that of a high-pitched sound, and *vice versâ;* and so also in the case of the percussion sound, if the number of higher tones preponderates greatly over that of the lower tones the general sound appears to be high in pitch, and *vice versâ.*

It has been said that when the chest wall is struck, the underlying air columns are set in vibration. This is due in great measure to the direct transmission of the impulse, but in some degree, at any rate, these vibrations seem to arise by sympathetic resonance. This demands a few words of explanation.

If a series of tuning-forks, of different pitch, be in turn sounded over the mouth of an empty jar, it will probably be found that the series contains one fork to which the air in the jar, so to speak, answers, which, when it is sounded, throws the air column into sympathetic vibration, so as to reproduce and strengthen its own note. If the air column be measured it will now be found that its length is exactly one-fourth the length of the sound-wave produced by the fork in question. This reproduction and reinforcement of the tone is termed resonance. In the same way, if a compound tone be sounded in the neighborhood of such an air column it will be set in sympathetic vibration if the sound-wave of the prime or any of the upper partial tones hap-

pens to bear the relation to the length of the column which has just been stated. Now, when the chest wall is percussed it vibrates, as I have already pointed out, and gives rise to a sound which is usually inaudible, and which, in any case, is of little importance. But underlying it there are numerous air columns, certain of which are of a suitable length to be set in sympathetic vibration by certain of the tones of which the sound of the thoracic wall is composed; so that the quality and general pitch of the intrathoracic note does to a certain extent depend upon the vibrations of the chest wall. The slight difference in pitch of the percussion sound during expiration and inspiration is thus to be explained. When the chest is in the condition of full inspiration its walls are tenser than during expiration, and, therefore, give a higher pitched note when percussed, which note is reproduced and strengthened by the resonance of the intrathoracic air columns, and thus raises the general pitch of the percussion sound.*

3. *Quality.*—The quality of a musical note depends upon the number and character of its upper partial tones. Enough has been said in the last pages to explain how this applies to the compound percussion sounds; and the various well-marked qualities which are to be met with clinically will be best discussed and explained in the next chapter when we come to deal with the practical aspects of percussion.

---

* The pitch, then, of the percussion sound at any given point depends upon the length of the air columns which are set in vibration, whether that vibration be produced by direct impulse or by sympathetic resonance; and the general pitch of the compound percussion sound depends upon whether the high or low-pitched notes are of larger number or greater intensity. But, as I have already said, the length of some at least of these columns is determined by limiting pulmonic septa. If the lung tissue become relaxed, these septa no longer limit the length of the air columns, which then extend back to the opposite wall of the chest, and consequently give a lower pitched note. Thus, as a whole, the percussion sound depends for its pitch upon three factors—(1) the tenseness of the chest wall; (2) the tenseness of the lung tissue; (3) the length of the underlying air columns.

CHAPTER XVIII.

Respiratory System—(*continued*).

PERCUSSION OF THE CHEST.

In the preceding chapter I have attempted to explain the theory on which the practice of percussion rests. It is now necessary to consider it in its clinical and practical aspects. And first, of the

Methods of Percussion.—There are two varieties of percussion, the immediate and the mediate.

Immediate percussion, or that in which the chest wall is struck directly with the finger, was the method originally employed. It is now almost completely discarded, the only exception being the percussion of the clavicles, which may with advantage be struck with the pulp of the forefinger before the percussion of the chest generally is commenced.

Mediate percussion, or that variety in which the finger or pleximeter is laid upon the chest wall and receives the stroke, is now almost universally employed.

As a general rule, it is probably best to use one or more of the fingers of the right hand to give the stroke, and to employ the fore or middle finger of the left hand as a pleximeter, applying its palmar surface firmly to that portion of the chest wall which we wish to percuss. For the right hand a percussion hammer may be substituted, and for a pleximeter we may employ an ivory or vulcanite plate of any of the numerous forms which are to be found in the shops of surgical instrument makers.

Whether the stroke be delivered by means of the bent finger or the hammer, it must be given from the wrist alone, and not from the shoulder or elbow, and the fingers or hammer must be raised from the pleximeter the moment the blow has been given, so as to allow of the free vibration of the chest. Skilful percussion with the fingers is very difficult to acquire, and requires long practice; but all students should learn it, for although hammer percussion is much easier, the physician may often be in circumstances when he is compelled to percuss without the aid of that instrument. Finger percussion is also much better suited to give

the feeling of resistance which, as will be presently shown, is often of considerable importance.

The patient, if a male, ought to be stripped to the waist, and if a female, only one thin and soft garment ought to be allowed to interpose between the chest and the finger or pleximeter.

It is of great importance that the chest should be percussed symmetrically, corresponding points on both sides being compared with one another, and it is necessary to see that the patient assumes no position of head, limbs, or trunk which will produce unequal muscular tension on either side.

As a rule, percussion need not be very forcible, though when the chest walls are thickened from the deposition of fat, or are dropsical, a strong blow may be necessary in order to produce a sufficiently audible note.

The Thoracic Percussion Note.—In the preceding chapter I have indicated the theoretical basis on which I believe the practice of percussion may safely be held to rest; but whatever their theoretical beliefs, most physicians will agree that the percussion sound depends mainly upon three factors—viz., (1) the thickness and tension of the chest wall, (2) the tension of the pulmonic tissue, and (3) the amount and disposition of the underlying air; and that it is to physical changes in these three factors that we must look for the cause of variations in the percussion note.

In the following pages we shall consider, firstly, the changes in the percussion note which occur as regards (*a.*) intensity, (*b.*) pitch, and (*c.*) quality (such as the tympanitic note, cracked-pot sounds, etc.); secondly, the feeling of resistance during percussion; thirdly, the topographical percussion of the lungs; and fourthly, the subject of phonometry.

The Intensity of the Percussion Sound.—As has been already said, the intensity of a simple pendular tone depends upon the amplitude of its vibrations; but in regard to the compound percussion sound, account must also be taken of the number of air columns which are thrown into vibration, and this depends upon the force of the stroke, upon the condition of the chest walls, and upon the volume of underlying air. Remembering that when two parts of the chest are being compared the force of the stroke must in each

case be equal, we may limit our attention to the two last factors.

1. *The Condition of the Chest Wall.*—When the thoracic wall is thickened by the deposition of fat, or by the transudation of serum into its interstices, the subjacent air is thrown with greater difficulty into vibration by the percussion stroke, and the resulting sound is deadened in passing through the thickened chest wall to reach the ear of the physician. The same diminution of the intensity of the sound occurs in health over those portions of the chest which are covered with thick muscle—for example, the scapular regions, and over the pectoralis major; and it must be borne in mind that in laborers in whom, from their occupation, the right pectoralis is considerably more developed than the left, the percussion note is less intense on the right side over that muscle than at a corresponding point on the left. Pleural effusions, also, have the same effect on the intensity of the note, as the layer of fluid prevents the free transmission of the percussion stroke. Such collections of fluid have, of course, a further influence on the note from the compression of the lung tissue which they occasion. The thickening of the pleuræ, which remains after an attack of pleurisy, tends to diminish the intensity of the percussion note, partly owing to the increased thickness of the chest wall thereby produced, but also, I think, from the manner in which the strong adhesions formed tend to contract, and so bind together the chest wall as seriously to interfere with its free vibration.

2. *The Amount of Air contained in the Chest.*—The intensity of the chest note is diminished whenever, from any cause, there is a serious diminution of the air contained in the chest. This may result either from compression of the lung tissue, so as to expel the air, such as takes place in cases of pleural effusion and of tumors pressing upon the lung, or from infiltration into the alveoli, such as occurs in pulmonary œdema, in the exudative stage of acute pneumonia, and in all the forms of chronic phthisis.

An *increase* in the intensity of the percussion sound is met with (1) where the chest walls are thin, in the young, the old, and in emaciated subjects; and (2) where the volume of air is increased, as is seen when the percussion note of full inspiration is compared with that of expiration; and further, in cases of emphysema, where the absolute volume of intrathoracic air is increased, not merely because many of

the pulmonary septa have disappeared and their place has been taken by air, but also on account of the permanent position of the thorax in the condition of full inspiration.

The Pitch of the Percussion Sound.—The pitch of a note depends upon the rapidity of the vibrations of which it is composed, and I have explained in the previous chapter how the term pitch may be more or less correctly applied to such a compound sound as that of percussion. The pitch of the thoracic note depends upon three factors—(1) the tension of the chest wall, (2) the tension of the lung tissue, and (3) the length of the underlying air columns; and I have already shown how these three conditions tend to modify the pitch of the note.

1. *The State of Tension of the Chest Wall.*—When a full inspiration is made, the tension of the chest wall increases, and consequently the percussion note tends to rise in pitch, although this tendency is to a certain extent counteracted by the increase of the volume of air in the lungs which then takes place. As a whole, however, the pitch during inspiration is higher than during expiration. In the same way, in pulmonary emphysema the percussion note is usually raised in pitch, owing in great measure to the increased tension of the thorax; and though the intensity of the note (as has been already said) is increased in these cases, I have frequently seen emphysema mistaken by the inexperienced for pulmonary consolidation, owing to the heightened pitch.

2. *The State of Tension of the Pulmonic Tissue.*—The increased tension of the lung tissue, during full inspiration, no doubt tends to heighten the pitch of the percussion note, along with the tension of the chest wall above mentioned. The results of relaxation of the lung tissue will be best described when we come to speak of the tympanitic note.

3. *The Length of the underlying Air Columns.*—Whenever the air-containing cavity lying under the point of percussion becomes more or less filled up, either as the result of effusion of fluid into the pleural cavity, or of effusion or exudation into the alveoli, such as take place in œdema, pneumonia, and in the various forms of phthisis, the air columns become shortened, and the percussion note rises in pitch. The same result follows where, from the deposition of new formations in the lung tissue, the air columns become broken up in their length. Within these limits fall the greater number of pathological conditions which are met

with in connection with the lungs. In each special case it is not difficult to see how the note becomes modified as regards its pitch.

In like manner is to be explained the change of note which occurs when we pass from the lungs to such solid organs as the liver and heart, and which enables us to map out their outlines in the manner already described. Take the liver for an example: As we percuss downwards in the mamillary line, we reach the upper margin of relative liver dulness—that point, namely, where the sound first becomes modified. It is here that the liver, lying in the hollow of the diaphragm, first begins to encroach upon the air space, filling it up from behind, and thereby shortening the air columns, and diminishing the volume of underlying air. The intensity of the note is thereby diminished, and its pitch rises, and these changes in the percussion note become more and more marked until we come to the upper limit of absolute liver dulness, where no lung tissue interposes itself between the liver and the chest wall, and the note, therefore, becomes absolutely dull. In the same way, the topographical percussion of other solid organs is to be explained.

Passing now to the consideration of certain changes in the quality of the percussion note, we come first, and most importantly, to

The Tympanitic Percussion Note.—This variety of chest note differs from that of health in that it approaches much more nearly to a pure musical tone—that is, its vibrations become much more regular. The great regularity of these vibrations Gerhard has shown by means of König's sensitive flame reflected in a rotating mirror. This variety of percussion note is found in perfection over the stomach when that viscus is moderately distended with air. If the stomach be removed from the body, both orifices ligatured, and then moderately distended with air, it will be found to afford a tympanitic note on percussion; but if the distension be continued, a point will soon be reached when the note loses that peculiar quality and becomes muffled. The reason of this is not far to seek. In the case of moderate distension, the gastric wall is not sufficiently tense to pass into vibration, and thus the sound results simply from the vibrations produced in the contained air; but when the

walls become tense from over-distension they also vibrate, and the tones so produced do not harmonize with those of the vibrating air, so that the combined sound is irregular in its vibrations, and therefore no longer tympanitic.

Similarly, if a lung be removed from the body and allowed to collapse, it will, when percussed, give a tympanitic note, which disappears when the lung is again distended with air to a point corresponding to the normal condition. The air in the collapsed lung vibrates as a whole, and the lung tissue is not sufficiently tense to admit either of its passing into vibration. or of the stronger septa breaking up the air columns so as to render the combined note irregular and non-tympanitic, as is the case when the lung is in a state of normal distension. It is to be noted that the pitch of the tympanitic note (which is very readily made out) gives a trustworthy indication of the size of the air cavity, and this is very important as a means of distinguishing the note of the stomach from that of the neighboring intestine.

Further illustrations of the tympanitic note in health are to be found when percussion is made on the cheek when the mouth is moderately distended with air, or over the trachea. The latter example is of especial value in that it shows another property of the percussion note—viz., that when the orifice of the cavity is narrowed or closed, the pitch of the note falls. If the trachea be percussed, first with the mouth open, and then with it shut, this lowering of the pitch will be readily detected, and it will be still more obvious if the nostrils be at the same time compressed.

From what has just been said, it will be seen that the tympanitic note may occur in the chest under the following pathological conditions:

1. Relaxation of lung tissue.
2. The presence of underlying air cavities.
3. Pulmonary consolidation, allowing the broncho-tracheal air column to be set in vibration.

1. *Relaxation of Lung Tissue.*—Just as when the lung is removed from the body, and allowed to collapse, it gives a tympanitic note, so when a similar retraction and relaxation of the pulmonic tissue takes place within the thorax, this variety of percussion note may be heard. This is best marked in cases of pleuritic effusion, which, gravitating to the lower portion of the cavity, floats up the lung and causes retraction of the upper portions. When the effusion

is small in amount this tympanitic note can only be detected over that portion of lung which lies immediately above the upper limit of the fluid, but when the effusion is considerable the whole upper lobe may be tympanitic on percussion.* Similarly, effusion into the alveoli (in pneumonia or œdema) may produce a like result. In the first stage of pneumonia the change in the note seems to be produced by relaxation, occasioned by the inflammatory congestion of the lung issue. Phthisical consolidation of the apices may also be accompanied with an obscurely tympanitic note over the neighboring portions of lung.

It is particularly to be observed that the pitch of the tympanitic note which occurs under the above conditions is not altered by shutting and opening the mouth.

2. *The Presence of underlying Air Cavities.*—When the pleural cavity becomes filled with air (pneumothorax) a typically tympanitic note results from percussion, provided that the distension be not too great. Its pitch is not altered by opening and closing the mouth. When the cavity contains in addition serum or pus (hydro- or pyo-pneumothorax) the note changes in pitch with the position of the patient, the fluid gravitating to the most dependent part in each instance, and so altering the lengths of the air columns.

Cavities in the lung tissue, when filled with air of sufficient size, smooth walled, and near to the thoracic wall, also give a tympanitic note, and as they communicate with a bronchus, the pitch of their note varies when the mouth is opened and closed. The position of the long axis of the cavity may also be ascertained, if it contain fluid as well as air, for the movements of the fluid occasioned by alterations in the position of the patient cause changes in the pitch of the note, just as in hydro-pneumothorax (Gerhardt). This change is extremely characteristic of cavities in the lung.

3. *Pulmonary Consolidation, allowing the Broncho-Tracheal Air Column to be set in Vibration.*—I have already alluded to the tracheal sound, which is characteristically tympanitic. In health, however, it is not possible to set in vibration the air column in the bronchi and trachea by percussing over the chest. If, however, the lung tissue be consolidated, the impulse of the percussion stroke may be transmitted to the

* When the effusion is excessive the lung tissue becomes compressed, and the tympanitic quality of the note is consequently lost.

bronchi, and in this way the tympanitic tracheal note of Williams may be produced. This note is almost always found on the left side—rarely on the right, and it is characteristic of it that the pitch is altered by opening and closing the mouth, but *not* by changes in the position of the patient.

Another note peculiar in quality, which must be mentioned, is the

Cracked-Pot Sound (*Bruit de Pot Fêlé*).—The peculiar quality of this sound is caused by the sudden expulsion of air from a cavity through a small opening in its walls, and it is well heard when the hands are pressed together and struck upon the knee, in a manner well known to schoolboys, so as to produce a noise closely resembling the rattling of coin. It derives the name (which Laennec first gave it) from the resemblance to the sound produced by striking a cracked jar.

The cracked-pot sound occurs under the following conditions:

1. *In Health.*—In children, when the glottis is narrowed, either during a fit of crying or when a sustained high-pitched note is being sung, percussion of the chest gives this variety of sound, the air being suddenly forced from the lung through the narrow glottis.*

2. *In Cases of Relaxation of Lung Tissue*, which I have already described as favoring the production of tympanitic percussion, the cracked-pot sound may sometimes be heard.

3. *In Cases of Thoracic Fistulæ* and pneumothorax, when the percussion stroke expels air through the fistula with a hissing sound.

4. *In Cases of Pulmonary Excavation.*—This is by far the most frequent cause of the cracked-pot sound, so much so that when consolidation of the lung tissue exists, the cracked-pot sound may be taken as strong evidence of the presence of a cavity.

The last change in quality which we shall here consider is that which is known as

---

* Also in adults, when the chest is very hairy, and a pleximeter is being used, the cracked-pot sound is apt to be produced. Owing to the instrument not being closely applied to the chest wall, a layer of air intervenes and a portion is forcibly expelled by the percussion stroke, producing the sound in question. Moistening the hair does away with this difficulty.

Amphoric Resonance.—This sound is similar to one produced by striking on the side of an empty jar or cask, and it owes its peculiar metallic quality to the high-pitched upper partial tones which it possesses, and which are caused by the reflection of the waves of sound from side to side of the closed cavity. These upper partial tones die away slowly.

When speaking of the tympanitic percussion sound, it was pointed out that when the stomach is over-distended with air that quality is lost, the note becoming hard and metallic. Amphoric resonance is then formed, the sound waves being again and again reflected from the tense walls of the viscus.

The conditions necessary for the production of amphoric resonance are that the air-containing cavity should be of considerable size and superficial, that its walls should be smooth and resistant, and that it should either be completely closed or should only communicate with the external air by means of a small opening.

As in the tympanitic note, so also in the amphoric sound, the pitch of the prime tone enables us roughly to estimate the size of the cavity in question.

In thoracic percussion this amphoric echo is met with in two conditions—over pulmonary cavities, and in pneumothorax. In both these cases it is best to combine auscultation with percussion, the physician listening with a stethoscope in the neighborhood of the cavity, while an assistant percusses. For percussion it is best to use a plexiimeter, and to strike it with some hard substance such as a coin, as the metallic note thus produced brings out by sympathetic resonance the high-pitched upper partials of the cavity.

Feeling of Resistance During Percussion.—The sound which the percussion of the chest affords is not the only sensation which is perceived by the physician in consequence of the stroke. There is further a sense of the degree to which the chest walls yield to the force of the blow.

This feeling of resistance may be dependent solely upon such changes in the chest wall as tend to increase its solidity (such as deposit of fat, thickening of the ribs, etc.), but if we expect these, it gives a trustworthy and sometimes exceedingly valuable indication of the comparative solidity of underlying organs. Whenever the lung becomes airless,

whether from exudation or compression, the resistance is increased; and still more is this the case when effusion of fluid has taken place into the pleura, and most of all over intrathoracic tumors.

Diminution of resistance is met with in cases of pulmonary emphysema, when well marked, and in pneumothorax.

Topographical and Regional Percussion.—The limits within which the pulmonary percussion note is heard are of importance, not only in determining the outline of neighboring solid organs, but as a guide to the physical condition of the lungs themselves.

The Apices.—The upper limit of the lung note corresponds to a line which, following at first the clavicular portion of the sternomastoid muscle, curves over to meet the anterior margin of the trapezius, and then passes downwards to the seventh cervical vertebra. This line rises on each side to a point about 1¼ to 2 inches (3 to 5 centimetres, according to Leitz) above the clavicle, being perhaps a trifle higher on the right side. In percussing the apices, care must be taken that the patient's head is not turned to either side, and that the direction as well as the force of the stroke is the same on each side. The shrinking of the apices, both vertically and transversely, is one of the first physical signs of incipient phthisis, and is therefore of considerable importance. In pulmonary emphysema, the limits above given may be overstepped to a considerable extent.

The anterior margins approach each other at the level of the second cartilage, being separated only by the anterior mediastinum, and continue downwards parallel to each other as far as the fourth rib, where the margin of the left lung curves outwards to follow the line of the absolute cardiac dulness, as already described; while that of the right lung continues vertically downwards as far as the sixth cartilage, where it joins the inferior margin.

The inferior margins are much affected by respiration. Their position during quiet respiration may be taken to be as follows:

The right lung—

At sternal border,	6th rib.
Parasternal line,	6th rib.
Mammillary line.	7th rib, upper border.
Axillary line,	8th rib.
Scapular line,	9th rib.

At vertebral column, . . . 11th rib.
The left lung—
Axillary line, ... 8th rib.
Scapular line. . . . . 9th rib.
At vertebral column, . . . 11th rib.

With forced respiration the inferior edges of the lungs rise and fall very considerably—to such an extent, indeed, that in the axillary line there may be a difference of over 3 inches between full expiration and full inspiration. In cases of emphysema, not only are the lower borders much depressed, but their movement during respiration is greatly interfered with.

The influence of emphysema, and other pathological conditions on the anterior borders of the lungs, has been already alluded to in connection with the percussion of the heart.

Regional Percussion.—The difference of the percussion sound at different parts of the healthy lung depends upon the condition of the chest wall, and upon the number and disposition of the air columns which radiate from the point struck.

Anteriorly.—The sound over the apices above the clavicles is clear, but not great in intensity. Below the clavicles the note falls somewhat in pitch, and grows in intensity until we come to the relative dulness of the heart on the left side (lower margin of third rib) and of the liver on the right (fourth inter-space, or fifth rib), when in both cases the sound rises in pitch and loses intensity, and does so more and more until the limit of absolute dulness of each solid organ is reached. The right lung is usually slightly duller than the left, owing to the greater development of muscle on the right side. Over the sternum the sound is clear, deep, and resounding, owing in part to the vibrations of that bone, but chiefly to the fact that the air in both lungs is set in vibration.

Posteriorly.—In percussing the thorax posteriorly, the patient should be made to cross his arms in front and bend forward. The note over the scapulæ is less clear than that at the lower portions of the back. The lung note can be heard as low down as the tenth or eleventh rib.

Laterally.—In the axillary regions the pulmonary note is intense and clear on both sides until the dulness of the liver is reached on the right side, and that of the spleen on the left.

CHAPTER XIX.

RESPIRATORY SYSTEM—(*continued*).

AUSCULTATION.

THE auscultation of the lungs may be performed with the aid of a stethoscope, or more simply by applying the ear to the thoracic wall. For obvious reasons, the former method is the pleasanter both to patient and to physician, and it possesses this further advantage that, by means of the stethoscope, any abnormal auscultatory phenomenon can be more distinctly localized than is possible if the immediate method be employed. The form of instrument is of comparatively little importance, provided that it fits the ear of the physician. The simple wooden stethoscope answers admirably for all ordinary cases, although sometimes the double instrument of Alison may be made use of with advantage.

The position of the patient is of considerable importance. Where there is a choice, probably the sitting posture is the most convenient, but whatever attitude be adopted it must be unconstrained. Of at least equal moment is the posture of the physician, which should be easy and comfortable. The chest of the patient should, if possible, be fully uncovered; but failing this, the intervening clothes must be thin, and all friction between them and the stethoscope sedulously avoided. The instrument should be firmly and accurately applied to the chest, and not till then should the physician apply his ear to the upper end, always remembering that the ear must be moved so as to suit the stethoscope, and not the stethoscope to suit the ear. Attending to these precautions, the whole chest must be carefully examined, corresponding points on the two sides being compared in the same manner as in percussion.

On auscultating the chest there is to be heard at most points a gentle "breezy" sound—the vesicular murmur—which resembles the sighing of wind among leaves, and the special character of which is readily learned by a little practice. It consists of two murmurs, the one corresponding to inspiration and the other to expiration, of which the first is about three times as long as the second, and is softer

and higher in pitch.* Not only do pathological changes in the lungs alter the ordinary respiratory murmur, but they often produce totally different sounds, to which in turn our attention must be directed. In ordinary clinical examination, then, the main points to be attended to in regard to auscultation are—

1. The relative duration of the expiratory and inspiratory murmurs.
2. The character or quality of the breathing sounds.
3. The presence or absence of adventitious sounds of various kinds.
4. The character of the vocal resonance (auscultation of the voice).

Vesicular Breathing.—In speaking of murmurs arising in the blood current, I have already pointed out that when a fluid streaming through a tube passes from a narrower into a wider portion, vibrations arise in the fluid owing to the friction of the molecules upon one another, which, if sufficiently rapid, give rise to an audible murmur. This is equally true with regard to gases. Now, in the air passages there are two points at which such an alteration in calibre is to be found—viz., at the glottis and at the point where the bronchioles enter the alveoli. The rush of air through the narrow glottis sets in vibration the air column contained in the trachea and bronchi, and a blowing murmur results—the tracheal or bronchial murmur—which will be described more fully hereafter; and in a similar manner a murmur arises as the air streams into the air cells. To a combination of these two murmurs, in which the latter predominates, it appears most reasonable to ascribe the formation of the normal vesicular breath sound.† Whatever be its origin, however, it may be safely held that, when vesicular breathing is heard, the pulmonary alveoli are fulfilling their function, and when it is absent, that that function is in more or less complete abeyance.

Vesicular breathing is to be heard more or less clearly

* The expiratory murmur is, however, not unfrequently inaudible in healthy persons.

† Baas, Gerhardt, and others, however, hold that the vesicular as well as the bronchial murmur arises solely at the glottis, and that the sound is in the former case modified by transmission through the lung tissue. The theory is attractive, but the proof offered seems inadequate, more especially as clinical facts point in the other direction.

over the whole pulmonary surface, but it varies in distinctness at different parts according to the thickness of the chest-wall and the volume of lung tissue underlying the stethoscope. From various causes vesicular breathing may be absent. Thus it may be replaced by bronchial breathing, or it may be inaudible, owing to the loudness of superadded sounds, or to the interposition of a tumor or of fluid between lung and chest-wall; or, finally, it may be absent owing to obstruction of a bronchus or to collapse of lung tissue.

The common modifications of vesicular breathing are as follows:

1. Harsh or puerile.
2. Jerky.
3. Prolongation of the expiratory murmur.
4. Systolic vesicular breathing.

(1.) *Harsh Vesicular Breathing.*—In children the normal vesicular breathing is clear, sharp, and loud, and this harsh or puerile breathing appears to depend in part upon the thinness of the chest-walls and the greater elasticity of the lung tissue.

In adults harsh or puerile breathing usually indicates a catarrhal condition of the bronchial mucous membrane. When heard over the lung tissue generally it is not, as a rule, of so much importance as when the harsh quality is only perceptible at one or both apices, which sign, when persistent, points strongly to incipient phthisis.

(2.) *Jerky Breathing.*—In nervous persons, and particularly in hysterical women, the inspiratory vesicular murmur is very apt to be broken into three or four distinct parts. This jerky breathing, which is heard over the whole lungs, disappears when the patient is told to take a deep inspiration, and is of no practical importance. But there is another variety of jerky breathing which does not so disappear, which is localized, and which is met with in incipient phthisis. This broken inspiration is a sign of considerable importance, and depends for its production upon some local obstruction to the entrance of air into the alveoli.

(3.) *Vesicular Breathing with Prolonged Expiration.*—It has been said that in healthy persons the expiratory murmur is frequently inaudible. When it is audible its duration is usually about one-third that of inspiration. When expiration exceeds this length we may conclude that either the lung tissue has lost its elasticity, or that there is some

obstruction to the escape of air. One or other of these two conditions is met with in almost every affection of the lungs, so that in pulmonary disease a prolongation of the expiratory murmur is almost universal.

(4.) *Systolic Vesicular Breathing.*—Often during inspiration the breathing may be heard to be momentarily strengthened during the cardiac systole. When the heart contracts, the neighboring portions of lung expand more rapidly to take the place formerly occupied by the heart in diastole, and thus the systolic strengthening of the inspiration occurs. Its presence has not as yet been shown to be of any diagnostic significance.

Bronchial Breathing.—The second great variety of respirtory murmur is that which is know as laryngeal, tubular, or bronchial. It can be heard in perfection when the stethoscope is placed over the larynx or trachea. Its peculiar character may be imitated by arranging the position of the mouth and tongue to utter the gutteral "Ch," and then breathing quietly out and in. The expiratory portion of the murmur is as long or longer than the inspiratory, and usually somewhat lower in pitch.

Bronchial breathing cannot be heard, in health, over the chest generally. Its area is confined to the larynx and trachea in the neck, and to the interscapular region close to the vertebral column* (from the seventh cervical to the third or fourth dorsal vertebra), opposite to the bifurcation of the trachea, where, however, its special character is not so well marked as in the former situations.

Mode of production of the Bronchial Respiratory Murmur.—The air passing in and out of the chest with the movements of respiration encounters at the glottis a considerable narrowing of the tube through which it is flowing, and in consequence vibrations arise in the immediate neighborhood of the narrow point, which are of sufficient rapidity to be audible as a murmur. Underlying this vibrating point, however, there is the air column contained in the trachea and bronchi, which is set in vibration by sympathetic resonance, and thus the glottis murmur is augmented and reinforced. It is in this manner, in all probability, that the bronchial murmur in healthy persons is produced. It can

* It is usually more distinct on the right side than on the left, owing to the greater calibre and more superficial position of the right bronchus.

readily be understood how, when the opening of the glottis is narrowed by such a pathological process as croup, the murmur is louder and higher in pitch.

To the vibrations at the glottis, and those of sympathetic resonance in the broncho-tracheal air column, other vibrations may, however, be added in consequence of pathological changes in the air passages. When the lumen of the trachea is narrowed, as the result of the pressure of a tumor, vibrations and a consequent murmur arise at the stenosed point, and are reinforced by the underlying air-column just as the glottis murmur is; and even when the mucous membrane of the trachea and bronchi becomes swollen and roughened by catarrhal processes, the character of the bronchial murmur changes, and it becomes harsh—the result, no doubt, of local vibrations.

The Bronchial Murmur in Disease.

As I have already said, the bronchial murmur is only audible in health over the larynx, trachea, and less distinctly between the shoulder-blades. It cannot be heard over the chest generally, partly because it is overpowered by the vesicular murmur, and partly because inflated lung tissue is a very bad conductor of sound. This murmur becomes audible, however, under two varieties of pathological conditions, as follows:

(1.) *When the lung tissue becomes condensed*—provided that the condensation is extensive, and lies at or close to the surface of the lung, and contains besides a large and unobstructed bronchus—the vesicular murmur disappears over the condensation, and the bronchial murmur is conducted to the surface and becomes audible. These conditions are fulfilled in the case of acute pneumonia (stage of hepatization), and in all the varieties of chronic phthisis. Bronchial breathing is therefore heard over hepatized lung, and wherever phthisical consolidation is of sufficient extent. It also occurs when the lung tissue is consolidated as the result of compression and collapse—as, for example, above the level of a pleuritic effusion; but it is not by any means always met with under such conditions, for the pressure of the effusion must be sufficient to cause collapse of the air-cells, and yet not sufficient to obliterate the bronchi.

(3.) *In pulmonary cavities.*—Bronchial breathing may be heard over vomicæ, provided that they are superficial, have

smooth walls, are surrounded by condensed tissue, and freely communicate by means of a bronchus with the air in the trachea. In certain cases it may be possible to judge roughly of the size of the cavity by the pitch of the bronchial murmur heard over it, since the air rushing into the cavity excites sympathetic resonance in it—that is, calls forth its special tone, which corresponds to the size of the resonating cavity, and this, if loudly enough heard, gives a guide to its size.

It is not, I think, desirable to subdivide bronchial breathing into a number of different varieties, as such a course only tends to cause confusion without apparently promoting any useful purpose. It may, however, be well to mention that many writers recognize a modification of bronchial respiration, which Lænnec named "cavernous breathing," in which the air appears to the ear of the auscultator to pass into a large hollow space. It is not, nevertheless, characteristic of the presence of a vomica.

There is, however, one special variety of bronchial breathing to which attention must be directed, viz :

Amphoric Respiration.

The peculiar character of this variety of bronchial breathing is perfectly reproduced by blowing into an empty jar or bottle, and its mode of origin is similar to that of the sound so obtained. Amphoric breathing occurs under two pathological conditions—(1) pulmonary excavation, and (2) pneumothorax, as follows :

(1.) *Pulmonary Excavation.*—The cavity must be of very considerable size, with smooth, firm walls, and must lie superficially. It must contain air, and must be in free communication with a bronchus. In such vomicæ the sonorous waves excited by the respiratory current* are reflected again and again from the smooth walls, and so come to have an amphoric character, the prime tone being comparatively low in pitch, and the upper partials high and ringing.

(2.) *Pneumothorax.*—When air escapes into the pleural sac and distends it, the lung tissue becomes compressed ; and

---

* As has been formerly explained, the sonorous waves formed in a pulmonary cavity by the respiratory air-current are the result of sympathetic resonance.

if this pressure be sufficient not only to drive the air out of the air-cells, but also to cause collapse of the bronchi, no amphoric breathing occurs. But if the fistula by which the air has entered becomes closed before the pressure has become sufficient to obstruct the bronchi, the bronchial respiration will be conducted to the immediate neighborhood of the large air cavity in the pleura, in which, by sympathetic resonance, sonorous vibrations will be excited. These vibrations, owing to the physical conditions met with in pneumothorax (smooth, firm walls, etc.), will have an amphoric character. If some quantity of serum or pus be present, along with air, in the pleural cavity, the pitch of the amphoric sound will vary according to the position of the patient, for the reason already mentioned.

Thus far I have described the two great classes of respiratory murmur, the bronchial and the vesicular. Between these two, however, their lies an intermediate variety, which may be called

Broncho-vesicular Breathing.

There may occasionally be heard a respiratory murmur which even the most practised ear cannot define as being either bronchial or vesicular. This murmur may be produced in healthy persons when they are directed to breathe superficially. In disease this variety of breathing is usually the result of partial blocking up of the bronchi leading to the part of the lung examined, or to the interposition of some badly conducting substance between the lung and the stethoscope, such as pleural effusion, tumor, or even œdema of the chest-wall.

This broncho-vesicular murmur is only of diagnostic value when localized in one particular part of the chest, particularly when it is confined to one apex. In the latter case it points to the probability of commencing phthisical change.

CHAPTER XX.

Respiratory System—(*Continued*).

ADVENTITIOUS SOUNDS ACCOMPANYING RESPIRATION.

In health the respiratory murmur is not accompanied by any other sound, but in the great majority of diseases of the lungs, at some part of their course, there become audible certain abnormal or adventitious sounds which are collectively known under the term râles. Inasmuch as certain of these râles give to the ear the impression of being caused by the bursting of air bubbles in a fluid, while others have a dry snoring or whistling character, they have been divided into two classes—moist and dry râles. Although this division is not scientifically accurate, some of the apparently moist sounds being in reality formed without the presence of fluid, and certain of the dry râles owing their production to the presence of a more or less viscid secretion, yet the division is clinically useful, and ought not, I think, to be discarded.

Physicians differ much in regard to the nomenclature of these râles, and as a rule they have been too minutely subdivided. For all practical purposes the following classification will be sufficient:

1. Moist râles—
   - (*a.*) Crepitation.
   - (*b.*) Fine bubbling râles.
   - (*c.*) Coarse bubbling râles.
2. Dry râles*—
   - (*a.*) Sonorous.
   - (*b.*) Sibilant.
3. Pleuritic friction.

Moist Râles.

Crepitant Râle.

The peculiar fine moist râle, which Laennec described under this name, has been compared to the sound produced by rubbing a lock of hair between the fingers close to the ear, or to the crepitation of salt when thrown upon the fire;

* Sometimes the term *rhonchus* is reserved for the dry sounds.

but, as Eichhorst points out, both these sounds are too coarse, and crepitation may be more closely imitated by firmly pressing the moistened thumb against the forefinger, and then suddenly separating the two surfaces, close to the ear.

Although crepitation is probably sometimes due to the bursting of fine bubbles in the very smallest bronchioles, it commonly arises from the sudden separation of the alveolar walls, which have become adherent either to each other or to a mass of viscid secretion in the air cell. It is typically met with in the first stage of pneumonia, of which it is a most important sign, and it also occurs in pulmonary collapse and œdema.*

Crepitation occurs almost invariably only during inspiration, and is usually limited to the latter part of it alone. The individual crepitations of which it is composed are characteristically uniform in size, and are unaffected by the act of coughing.

Occasionally in health a momentary crepitation may be heard, usually at the lower posterior border of the lung, but sometimes also at the apex, when a deep inspiration is made, more especially when the patient has been lying on his back, and the respiration has been very quiet for some hours. A knowledge of this fact will prevent any mistake.

Fine bubbling râles.

Coarse bubbling râles.

These two varieties of râle being closely associated, they may conveniently be considered together. The difference in the size of the bubbles in each case depends somewhat upon the quantity and quality of the fluid in which they originate, but chiefly upon the size of the space. The finer bubbling râles arise for the most part in the smaller bronchi, the coarser in the large bronchi, in the trachea, or in pulmonary cavities. In the great majority of cases in which these bubbling râles are heard, they vary in size, and are therefore spoken of as irregular, in contradistinction to the regular fine crepitant râle which has just been described.

Arising in fluid, as these bubbling râles do, we would naturally expect that they would be found most abundantly in the lower portions of the lung—the fluid obeying the law

---

* In œdema bubbling râles are superadded, owing to the bronchi being filled with fluid, and hence the uniformity of the crepitation is lost. The sound is thus easily distinguished from the crepitant râle of pneumonia.

of gravity—and this is generally the case, the base of the lung posteriorly being their most common seat. When, on the contrary, they are heard most abundantly at the apices, and still more when they are exclusively met with there and persist for some time, the condition is one which must be looked upon with considerable gravity, pointing as it does to a local cause, which in the majority of cases is some form of pulmonary phthisis.

The finer bubbling râles in the smaller bronchi occur chiefly at the height of inspiration and the beginning of expiration, while coarse bubbling may be heard both during expiration and inspiration, being then continuous. In both cases a severe fit of coughing may remove the râles for the time. Their amount and intensity depend upon the quantity of fluid, the nearness of the bubbling to the surface, and the strength of the respiration.

In so far as the properties of these bubbling râles, which have as yet been described, go, their presence only informs us that the air current encounters fluid in the respiratory passages, through which it bubbles. We now come to certain qualities in the tone of these râles which give an indication of the condition of the surrounding pulmonary tissue. If the lung tissue around the point at which the bubbling is taking place is consolidated, the râles assume a clear musical high-pitched quality, and are termed *resonant.* Whenever such râles are heard we may conclude, with safety, that consolidation is present (although their absence does not permit of the exclusion of such a condition), and in fact resonant bubbling has a significance exactly similar to that of bronchial breathing. When the râles occur in a large cavity with smooth walls and near to the surface of the lung, they assume a peculiarly clear metallic character—the *metallic tinkling** of Laennec. These râles are very musical, and have high pitch which can readily be determined, and in regard to their physical cause and the conditions under which they occur, they stand in close relation to amphoric breathing and resonance. Similar resonant râles may be heard over large air cavities which lie in close proximity to the lungs, such as a pneumothorax, or even the stomach or intestine when distended with air. In

* This tinkling was supposed to be sometimes produced by the dropping of fluid from the roof of a cavity, but this manner of production would seem to be exceedingly doubtful.

such cases it is not necessary that the râles arise in pulmonary cavities; they may originate simply in the bronchi, and the neighboring air cavity may act as a resonator, reproducing and intensifying the sound.

Dry Râles are produced in the air passages by any pathological process which narrows their lumen, the most common being the accumulation of viscid secretion and the swelling of the mucous membrane. When they arise in the larger bronchi they are low-pitched and snoring (sonorous râles), when in the smaller tubes they have a whistling character (sibilant râles). Both varieties occur chiefly during inspiration, the snoring râles at its commencement, the sibilant not till towards its termination.

Both these varieties of dry râles occur in cases of bronchial catarrh, whether acute or chronic, primary or secondary, and according as they are sonorous or sibilant we may infer that the larger or the smaller bronchial tubes are affected.

The presence of pulmonary consolidation round the point at which these râles occur imparts to them a ringing musical character, but as their quality is in any case musical, this change has not anything like the diagnostic value which it possesses in the case of moist râles.

Pleuritic Friction.—The gliding of one pleural surface over the other, which occurs normally with each respiration, is accomplished without any sound; but when, as the result of pleurisy, the surfaces become rough and uneven, sound of friction becomes audible. This sound varies from the lightest rubbing, only perceptible with difficulty, to loud creaking, which can readily be made out on palpation, and which the patient himself both feels and hears. The sound is usually broken up into portions of greater and less intensity, and while it is sometimes audible throughout the whole of both respiratory phases, it is usually limited to the latter portion of inspiration. In cases of pleurisy the friction sound becomes audible whenever the process has advanced sufficiently far to cause considerable roughness of the pleural surfaces, and it of course disappears when those surfaces are separated by effusion, to reappear when absorption of the fluid has taken place. Although the friction sound may sometimes be audible over a great part of the lung, it is usually limited to a small area, and occurs

most frequently in the axillary region. When the friction sound is heard at the apex of the lung it points with great probability to phthisis.

With regard to differential diagnosis, the pleuritic friction-sound is sometimes closely simulated by râles in the air passages.

Attention to the following points will usually suffice to distinguish them:

Râles.	*Friction.*
Modified by coughing.	Not modified by coughing.
Not affected by pressure of the stethoscope.	Intensified by pressure of stethoscope.
Usually heard over wide area.	Usually localized.

From pericardial friction the sound of pleuritic friction is easily distinguished by making the patient cease breathing, when the latter will disappear and the former continue.

Auscultation of the Voice.—In a former chapter, the fremitus, or vibration of the chest-walls, produced by the act of speaking, has been described. As regards its causation and the various pathological conditions under which it is enfeebled or intensified, the resonance of the voice closely corresponds to the vocal fremitus.

When the stethoscope is applied to the chest while the patient speaks, only a soft indistinct murmur is to be heard, provided that the lung is healthy. Over the larynx and trachea, however, this vocal resonance is much intensified, and it is almost as though the words were spoken into the opening of the stethoscope. This intensification is termed *bronchophony.**

Before speaking of changes in the vocal resonance produced by pathological conditions connected with the lung, it may be as well to repeat what was said in connection with vocal fremitus—viz., that the vibrations of the voice over the thoracic parieties, audible as well as perceptible to palpation, depend for their intensity upon the loudness and depth of pitch of the voice, and upon the thickness of the chest-wall; that the vocal resonance (like the correspond-

* The highest development of bronchophony was termed pectoriloquy by Laennec, but there is no fundamental difference between the two, and there seems to be no good reason for adding an additional term to the auscultatory nomenclature, which is already sufficiently complicated.

ing fremitus) is more distinct in men than in women; and that it is almost invariably louder on the right side than on the left, owing to the larger calibre of the right bronchus.

Bearing these points in mind, we may now consider the changes in the vocal resonance which result from pulmonary disease.

Enfeeblement of the Vocal Resonance.—The vocal resonance is diminished when the lung is separated from the chest-wall by collections of liquid* or air in the pleural cavity, and or when the bronchi leading to the part of the lung in question have become blocked up with secretion.

Intensification of the Vocal Resonance (*Bronchophony*).—As has been already said, bronchophony occurs normally over the larynx and trachea down to the bifurcation of the latter in the interscapular region. When bronchophony occurs at other points of the chest it is pathological, and it then owes its origin to consolidation of lung-tissue, and the consequent better conduction of the vocal vibrations to the chest-wall. Bronchophony thus arises, along with bronchial respiration, in all diseases which lead to condensation—for example, in acute pneumonia, and in all the forms of phthisis. It is particularly noticeable over pulmonary vomicæ, the resonance of the air in the cavity adding to the intensity of the vocal resonance, and imparting to it in addition a peculiar metallic character. As a whole, it may be taken that bronchophony has an exactly similar significance to bronchial respiration.

It has been said that pleural effusions diminish or even suppress the vocal resonance; but this is not always the case. Baccelli pointed out, in 1875, that the resonance of the whispered voice was often heard very clearly over pleural effusion. This *pectoriloquie aphonique* he held to occur only when the fluid was homogeneous (serous effusion), and was not present when the effusion was heterogeneous (pus). There can be no doubt that this sign is very frequently present in such cases, but recent observations† have failed to confirm its value in so far as the discrimination between serous and purulent effusions is concerned.

Under certain conditions, bronchophonic vocal resonance assumes a very peculiar nasal quality, resembling the noise

---

* With the exceptions to be presently mentioned.

† Dr. Douglas Powell, Tr. International Med. Congress, 1881.

produced by speaking against a comb covered with paper, and which, from its supposed resemblance to the bleating of a goat, Skoda termed

Aegophony.—This variety of bronchophony is most commonly met with in cases of pleuritic effusion, near the upper margin of the fluid, and usually close to the lower angle of the scapula. As to the exact manner of its causation there is some doubt, but most observers are agreed that it depends upon compression and partial obstruction of the bronchi. Its diagnostic value does not materially differ from that of ordinary bronchophony.

Hippocratic Succussion.—We must, in conclusion, refer briefly to this sign, which was described by Hippocrates, and which, although rarely met with, is of considerable interest.

If, in cases of pyo-pneumothorax, the ear be applied to the chest and the patient shaken, a ringing splashing sound may be heard, which is the sign in question. The splashing noise becomes intensified by the resonating air cavity above the fluid in the way I have already described. This succussion sound may also be heard when there is a very large excavation in the lung tissue partially filled with fluid.

CHAPTER XXI.

Integumentary System.

The study of the affections of the skin is of great importance to the physician, not merely on account of the frequency of their occurrence, the distressing severity of their symptoms, and the deformities which they leave behind them in their course, but also because they are frequently symptomatic of the general condition of the system, mirroring forth with fidelity not a few grave systemic diseases, and still more often the many slighter disorders which it is important to recognize and to check at their outset, and of which the physician may have no other indication. Among the

---

* Dr. Douglas Powell, Tr. International Med. Congress, 1881.

serious diseases of which the condition of the skin gives evidence, it is hardly necessary to mention syphilis, scrofula, as well as all the members of the important group of exanthemata. Then, again, it is well known that errors in diet, and disorders of the digestive functions generally, are apt to cause various forms of skin disease (urticaria, acne, etc.), and without going further into detail in illustration of this point, it may finally be mentioned that many uterine affections, and even pregnancy itself may be accompanied with blotches on the skin (chloasma uterinum), which in certain circumstances may be a symptom of not a little importance.

Subjective Symptoms are of comparatively little diagnostic importance in cases of skin disease, although to the patient they are often very distressing. Sensations of heat attend all the inflammatory processes in the skin. Hyperæsthesia and anæsthesia are met with in the various cutaneous neuroses, and shooting pain is a prominent symptom of herpes zoster, frequently preceding the eruption, and persisting for some time after its disappearance. But of all the subjective symptoms of skin disease, the most distressing is itching. It is very common as a result of the presence of parasites, but frequently occurs independently of such agents in cases of eczema and of pruritus.

Objective Symptoms.—A patient suffering from skin disease ought to be examined in a well-lighted and warm room, preferably by daylight, and if a male the whole surface of the body ought to be viewed by the physician if the case is at all important.

The general condition of the skin as to color and moisture, the deposit of subcutaneous fat, and the presence of œdema and of emphysema are points which have been already considered in Chapter I., and need not be again referred to.

There only remain, therefore, for our consideration, the various eruptions which occur on the skin.

Eruptions.

In considering skin eruptions, there are four main points to which attention should be directed, and under which the facts observed may be satisfactorily classified.

1. *The Distribution and Configuration of the Eruption.*

When the whole surface of the body is covered, the eruption is said to be *universal;* when it is irregularly scattered over various parts of the body, it is said to be *diffused.* The configuration of the individual lesions is commonly defined by such terms as *punctate* when of the size of pin-heads, *guttate* when resembling drops of water, *nummular* when of the size of coin, etc.

The distribution of the lesion is often of considerable diagnostic importance. For example, psoriasis is usually only found on the extensor aspect of the limbs, whereas the macular, papular, and squamous syphilides, when they appear on the limbs, are seen on the flexor surfaces. Then, again, certain lesions, as herpes zoster, follow the course of nerves. Lupus is usually found on the face, erythema nodosum on the leg, seborrhæa sicca on the scalp, acne on the face and back, and the scabies insect selects for its burrows by preference the skin between the fingers.

2. *The Elements of the Skin Involved.*

Careful inspection of the skin will inform the physician as to the condition of the epidermis, the hair, the orifices of the hair follicles, the sebaceous and the sweat glands. Palpation of the skin will further give information regarding the condition of the true skin, whether it be infiltrated or not. Pressure with the finger on a pigmented spot will show whether the coloration is due to hemorrhage or to hyperæmia, for in the latter case the color will disappear on pressure. Further, if the skin be covered with crusts, the removal of these by means of the finger will display the condition of true skin, which in cases of eczema, for example, may be found to be moist, in seborrhæa dry, and in psoriasis bleeding. We have further in the diagnosis of skin cases to rely upon the evidence afforded by the microscopic examination of the hair, crusts, etc., as will be more particularly pointed out when we come to speak of the ætiology of such affections.

3. *The Type of the Eruption.*

The very numerous forms assumed by skin eruptions may be defined and described as follows:

(*a.*) *Macules* (*Maculæ*).—These consist in morbid changes

in the color of the skin, which are circumscribed, and do not involve the whole cutaneous surface, and which are neither elevated above nor depressed below the surface of the skin. Such macules may arise in very various ways. Sometimes, as in Erythema fugax, they are occasioned by hyperæmia, and then the color disappears on pressure; sometimes by hemorrhage, as in purpura; by increase or decrease of the normal pigment; by exudations into the tissues of the true skin, as in syphilides; and, finally, they sometimes arise from increase in the size and number of the blood-vessels, as in nævi.

(*b.*) *Papules* (*Papulæ*) are small firm elevations above the surface of the skin, varying in color, and arising in very different ways. The simplest form of papule is seen in the *cutis anserina* or goose-skin, due to the contraction of the muscles of the skin. Pathologically, papules form as the result of hypertrophy of the papillæ (ichthyosis), of cell proliferation in these structures (lupus, syphilis), or of inflammation of and consequent exudation into these papillæ, as in eczema papulosum. Extravasation of blood into the skin may give rise to papules, as is seen in purpura papulosa. Papules may also be formed in connection with the sebaceous glands (milium, comedo, acne), or by accumulation of epidermic cells round the hair follicles, as in lichen pilaris.

(*c.*) *Tubercules* (*tubercula*) are simply exaggerated papules. They are usually occasioned by cell proliferation, and occur as the result of syphilis, carcinoma, leprosy, etc.

(*d.*) *Tumors* (*phymata*) hardly require a definiton here. They may be of considerable size, even as large as a child's head. Examples are seen in molluscum, and in the various cystic growths met with in connection with the skin.

(*e.*) *Wheals* (*pomphi*) are flat, irregularly-shaped, firm, elevations on the skin, pale in the centre, red at the edges, and which are very fugitive. Wheals are typically seen as the result of the sting of the nettle, and in urticaria. They result from sudden effusion of the serum into the papillæ, and swelling of the cells of the rete malpighii, produced probably by vasomotor changes.

(*f.*) *Vesicles* (*vesiculæ*) are small rounded elevations of the cuticle, varying in size up to that of a split pea, containing serous, sero-purulent, or bloody fluid, and either lying between the mucous and horny layers of the epidermis, or

in connection with the hair follicles, or with the sebaceous or sweat glands.

(*g.*) *Blebs* (*bullæ*) only differ from vesicles in point of size, being larger than a split pea.

(*h.*) *Pustules* (*pustulæ*) are elevations of the epidermis, similar in shape to vesicles and blebs, but containing pus. They are sometimes found in substance of the true skin (boils), in connection with hair follicles (as in sycosis), or in sebaceous glands (acne), or between the mucous and horny layers of the epidermis, as in smallpox. Pustules usually dry up (with or without bursting) into yellow or brownish crusts, and very often leave permanent cicatrices, if the tissues of the true skin have been involved.

Thus far, we have been considering what are called the *primary* lesions, and we now pass to those which are secondary.

(*i.*) *Excoriations* are breaches of the continuity of the skin, produced most usually by the patient's nails. They give an indication of the amount of itching which is present. When lice are present (phthiriasis), the marks of the scratches are long and straight; in pruritus, they are short and irregular; and in scabies, small and round.

(*j.*) *Scales* (*squamiæ*) are portions of epidermis which have become separated by diseased processes in the skin. The deeper and more severe the inflammation, the more marked is the desquamation. The scales may be thrown off as fine branlike particles (as in prurigo, pityriasis, measles, etc.), or as thin flakes or thick plates (in psoriasis, and eczema); or the epidermal layer to be thrown off may, as in scarlatina, separate as a whole, forming a more or less perfect cast of the fingers, or even of the whole hand.

(*k.*) *Crusts* are formed by the drying up of the products of skin disease, serum, pus, blood, etc. When chiefly composed of pus, they have a greenish color; when mixed with blood, the crusts are brown or black. The firmest and hardest crusts are those met with in syphilitic processes (rupia), when they often assume a form closely resembling that of a limpet shell. The crusts of favus are yellow and cup-shaped.

(*l.*) *Fissures* (*rhagades*) in the skin may involve the epidermis alone, or both the epidermis and the true skin; or they may be seated in mucous membrane. They are usually found where the skin is normally furrowed—as, for example, on the palms of the hands and soles of the feet; at the

angles of the mouth ; where the upper lip and nose join ; at the elbows and knees ; at the anus, and in other similar situations. Fissures are found in cases of chronic eczema and inveterate psoriasis, in syphilis, and in scleroderma.

(*m.*) *Ulcers* are chiefly within the domain of surgery. Their size, depth, shape, situation, and general condition should be noted.

(*n.*) *Cicatrices* follow all diseases or injuries of the skin which involve loss of substance. The character of the scar is not indicative of the preceding disease ; but sometimes the number or seat of the cicatrices may afford some indication of their cause.

4. *The Etiology of the Eruption.*

Skin eruptions are much influenced by the age and sex of the patient, by the season of the year, and by climate. On these points, and on the heredity of many such diseases, we need not now dwell. Very frequently skin affections are the result of constitutional diseases, as, for example, purpura, scrofula, rickets, all the acute exanthemata, diabetes, etc. We have further to note that diseases of particlar organs often give rise to skin eruptions. Disorder of digestion from improper diet (shell-fish, for example), or other cause, is frequently followed by urticaria and acne. In valvular disease of the heart, œdema and small hemorrhagic extravasations (petechiæ) frequently occur. Bright's disease is often accompanied by pruritus, and sometimes by eczema. Many other instances, too numerous to mention here, will occur to the reader in which diseases of the different internal organs are accompanied by skin eruptions which are more or less characteristic.

A class of eruptions with which it is very important that the physician should be familiar are those which result from internal and external use of certain medicines.

All counter-irritants—such as croton oil, mustard, cantharides, tartar-emetic, iodine, turpentine, arnica, etc.—give rise to various forms of dermatitis; as do also the various aniline colors with which stockings are sometimes dyed.

The internal administration of medicines is occasionally followed by skin eruptions, a result which is most frequently due to some peculiar idiosyncrasy of the patient. Among these may be mentioned the acne pustules which follow the use of the bromides, the erythema (or even eczema) of the

iodides, and the scarlatina-like efflorescence of chloral. Very characteristic of atropia-poisoning are the bright erythematous patches which appear on the chest and neck. Morphia sometimes gives rise to an erythematous eruption resembling that of scarlatina, and the administration of quinine is occasionally followed by a rash of the same description. The eruption of copaiba usually shows itself upon the extremities as a bright papular efflorescence, which is generally very itchy.

Among the causes acting locally in the production of skin eruptions may be mentioned (in addition to the external applications just noticed) the following: Continued exposure to the heat of a strong fire is apt to give rise—in furnacemen and cooks, for example—to eczema. Those who work in acids or alkalies, and especially in aniline dyes, suffer much from eczema. Even the long soaking of the hands and arms in hot water and soap produces in washerwomen a hardened, fissured, and even eczematous condition of the skin of these parts. The most important local cause of skin eruptions is, however, undoubtedly to be found in the irritation set up by the various parasites which infest the skin and hair. The diagnosis in such cases is closely bound up with the etiology, and they must be considered together. The parasites which affect the skin belong to both the animal and vegetable kingdoms. The most important of these are the following:

Vegetable Parasites.

1. *Achorion Schönleinii.*—This parasite gives rise to the disease known as tinea favosa, or favus. While it occasionally attacks the nails, it is most usually found upon the scalp, where it gives rise to the formation of light-yellow, dry, cupped crusts. The hair follicle and hair are first attacked, and then the parasite spreads itself upon the surface of the skin. When a part of one of these crusts is examined with the microscope, after having been soaked in water, and treated with acetic acid or an alkali, the parasite (fig. 6) is readily recognized. It consists (1) of spores; (2) of slightly elongated elements, which are usually united in rows; and (3) of mycelium, which is made up of long, branching, transparent filaments, which may or may not contain spores in their interior. In the favus crust there

are always to be found, in addition, numerous micrococci and bacteria.

2. *Trichophyton.*—This parasite produces three forms of skin disease—tinea circinata, or ringworm of the body; tinea tonsurans, or ringworm of the scalp; and probably tinea sycosis, or sycosis parasitica, a similar affection of the beard and other hairy portions of the face. On the body, the trichophyton gives rise to considerable irritation of the

FIG. 6.—Achorion Schönleinii (Duhring).

skin, which results in the formation of circular circumscribed patches of various size, slightly elevated above the level of the skin, of a dull red color, and usually covered with small branny scales, while round the edges there may be found vesicles, and sometimes even pustules.

On the scalp, ringworm shows itself as one or more circumscribed patches of a grayish or slightly ruddy color. The hair of the affected parts is short, lustreless, easily drawn out, breaks readily, and the extremities are ragged

and uneven. The skin is covered with numerous thin white scales, and occasionally with crusts.

On the beard and upper lip, the parasitic form of sycosis —which is probably caused by the trichophyton—at first exhibits characters closely resembling those of ringworm of the scalp. As the disease advances, however, the skin and deeper parts become inflamed and indurated, and, as a

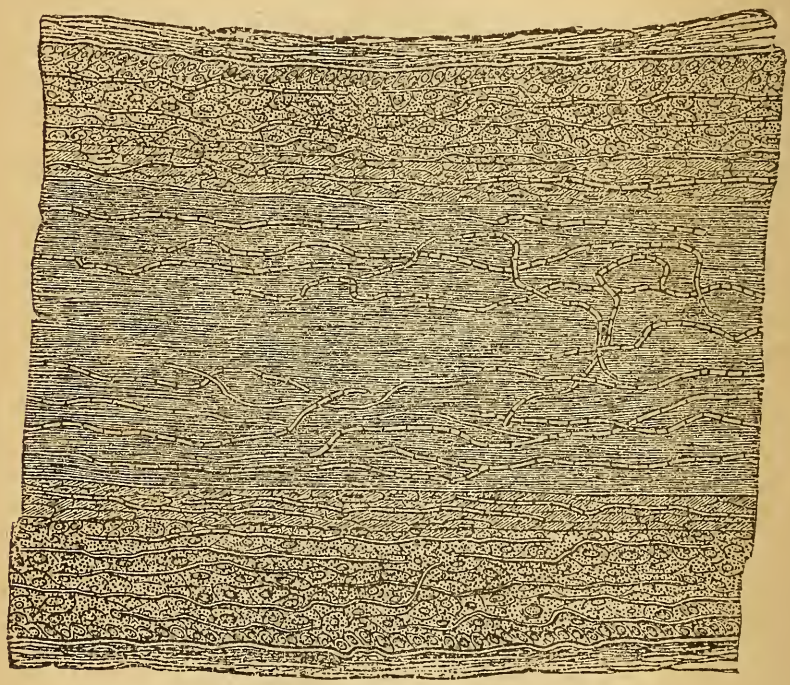

FIG. 7.—Hair affected with Tinea Tousurans (Neumann).

consequence, the affected portions become covered with characteristic tubercular elevations, and pustules occupy the hair follicles.

In all these situations the trichophyton presents similar microscopic appearances. In the case of tinea circinata, a few of the scales should be scraped off the patch with a penknife, laid on a microscopic slide and examined, after the addition of a dilute solution of carbonate of potassium. In the other forms, a diseased hair should be extracted and examined, after the addition of liquor potassæ or chloro-

form. Whether in the hair (as in fig. 7) or spread over the surface of the skin, the parasite will be found to consist of long, slender, jointed filaments (mycelium), together with small, round, highly refractive spores. The latter are most abundant in ringworm of the scalp, infiltrating densely the hair bulb, while the mycelium spreads up the shaft of the hair.*

3. *Microsporon Furfur* is the parasite which gives rise to pityriasis versicolor. This disease is characterized by the presence on the skin (usually of the back and chest) of variously-sized pale yellowish-brown or reddish patches covered with fine powdery scales. It is most frequently met with in those suffering from wasting diseases. The microsporon furfur consists of spores and mycelium. The spores are small, round or oval, highly refractive bodies, which tend to arrange themselves in groups in a manner which is very characteristic of this parasite. The mycelium consists of fine curved filaments which are usually short and are jointed together, forming a close network. In their interior spores are generally to be seen.

This parasite is very readily detected by means of the microscope. A few of the scales on the surface of the patch should be scraped off with the penknife, placed upon a cover-glass, and treated with liq. potassæ.†

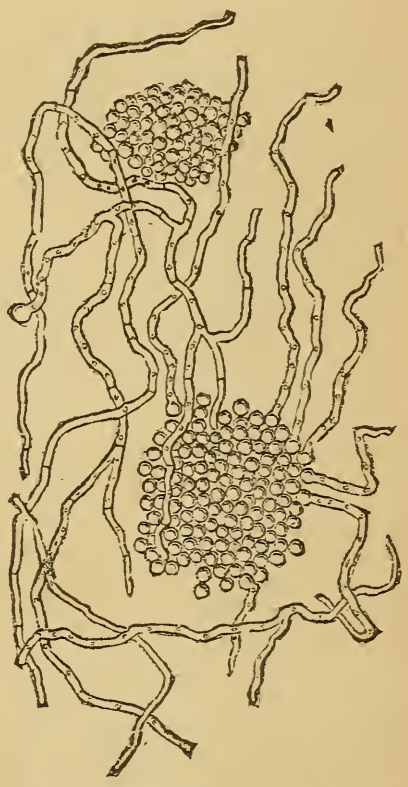

Fig. 8.—Microsporon Furfur (Neumann).

* It is very probable that a form of eczema which affects the inner surfaces of the thighs (eczema marginatum) also owes its origin to the presence of the trichophyton.

† The patches of baldness of alopecia areata are by some supposed to be parasitic, and to depend on the presence of the microsporon audoini, but there is considerable uncertainty on this point.

Animal Parasites.

1. *Sarcoptes Scabiei.*—The skin disease—scabies, or the itch—which is caused by presence of this insect, consists of papules, vesicles, pustules, excoriations, fissures, crusts; in short, an eczema, in the neighborhood of the burrows in which the insects lie. Scabies is usually found between the fingers, but may spread over the body generally, the insect selecting, however, localities where the skin is soft and thin. It is the commonest of all skin diseases, and is very contagious.

Fig. 9. Sarcoptes Scabiei (Neumen).

The female insect, which can be seen in fig. 9 lying at the end of its burrow, has an oval body, marked with fine undulating lines, a small oval head, and possesses eight legs (the four front legs being furnished with suckers) and numerous bristles. The male insect does not give rise to the eruption, and is but seldom met with. The female, after being impregnated, bores through the horny layer of the cuticle perpendicularly, and then forms a burrow which runs horizontally in the mucous layer of the epidermis. This burrow, or cuniculus, can often be seen by the naked eye as a whitish elevated line terminating in a minute speck, which is the insect. The cuniculus becomes filled with eggs, of which the insect lays about ten to fifteen, and with small black specks of excrement. The eggs are hatched in about ten days.

With a little trouble the itch insect can usually be se-

cured, and the diagnosis thereby rendered certain. A needle should be introduced into the burrow, which the insect will generally seize hold of, and on the point of which it may be removed.

2. *Pediculus.*—Three varieties of pediculi are met with on the human body, giving rise to the disease termed phthiriasis—the pediculus capitis, pediculus corporis, and pediculus pubis.

The head louse is an elongated ovalish insect of a grayish hue. From its head spring two antennæ, each consisting of five parts, and to the thorax are articulated six legs armed with strong claws. The oval whitish eggs are firmly fastened to the hairs of the patient's head by means of a viscid glue-like material. The irritation and itching of the scalp and the consequent scratching give rise to a severe eczematous condition, in which the serous, sanguineous, and purulent exudations matt together the hairs, and almost invariably cause enlargement of the neighboring lymphatic glands.

The pediculus corporis, or pediculus vestimenti, as the insect is sometimes termed, resembles closely the head louse in structure, but is somewhat larger. The cutaneous lesions consist chiefly in long scratch marks, crusts, and papules, along with the minute red points where the insect has bitten.

The pediculus pubis, or crab-louse, is smaller and more rounded than either of the other species. It infests chiefly the pubic region, and usually gives rise to considerable irritation.

3. *Pulex Irritans*, the common flea, need hardly be mentioned here, were it not that flea-bites are occasionlly mistaken for purpuric spots. Round the bite, however, there will be seen to be a hyperæmic areola, which is not met with in purpura.

4. *Demodex Folliculorum* is a harmless, worm-like parasite, which inhabits the sebaceous follicles of the skin of the face. If the contents of a prominent follicle be squeezed out and examined with the microscope, the demodex will be found.

CHAPTER XXII.

Urinary System.

SUBJECTIVE SYMPTOMS.

Before proceeding to the consideration of the various changes met with in the urine in disease, which must always rank as the most important sign of urinary disorders, it may be well to note certain subjective symptoms which occur in such cases, and which often give very valuable indications.

Pain may be felt at different portions of the urinary tract, as follows :

1. *At the end of the Penis.*—In calculus of the bladder pain is felt after micturition, because the rough stone then comes in contact with the bladder wall ; it is referred chiefly to the extremity of the penis, and is increased by any sudden movement. In prostatitis, also, pain occurs after passing water, the bladder then contracting on the tender prostate. In women there is often severe pain felt during micturition at the orifice of the urethra, owing to the presence there of a small vascular growth.

2. *In the course of the Urethra.*—When the urethral canal is narrowed by stricture, pain is felt at the constricted point during micturition. In urethritis, also, the pain during the passing of water is referred to the urethra. When the urine is highly acid, concentrated, or contains gravel, urethral pain also occurs during micturition.

3. *Over the Bladder in the Supra-Pubic Region.*—This is the common seat of the pain of cystitis, which, it is to be observed, occurs before micturition, and is relieved by that act. In acute cases pain may also be felt deep in the perineum.

4. *In the Loins.*—In cases of pyelitis,and of renal calculus there is usually dull aching pain over the loins, which is increased on pressure, and which in the latter disease occasionally passes into violent paroxysms, the pain shooting down the ureters to the testicle and inside of the thigh.

Frequency of Micturition.—Wherever the urine is large in

quantity, as in diabetes and the waxy form of Bright's disease, for example, there is frequency in micturition. This symptom, however, also occurs in very many other urinary disorders. In all inflammatory conditions of the prostate and bladder, in pyelitis, and nephritis, in calculus of the bladder or kidneys, the urine is frequently voided. It is particularly to be noticed that in the cirrhotic or contracting form of Bright's disease, and in hypertrophy of the prostate, the calls to micturate are frequent, and occur chiefly during the night.

The Examination of the Urine.

In such a work as this it is of course quite impossible to give anything like an exhaustive account of the many changes which take place in the urine in health and disease, or of the various methods of analysis which have been applied to that secretion. All that I shall attempt to do will be to enumerate the more ordinary and clinically significant changes which occur, and the simpler methods of analysis, such as may be carried out by the physician, excluding those which require the more complicated apparatus of a chemical laboratory.

In the present chapter we shall consider the general condition of the urine as to (1) quantity, (2) color and transparency, (3) odor, (4) specific gravity, and (5) reaction.

1. **Quantity of the Urine.**—While varying according to the quantity of the fluid drunk, the amount of the pulmonary and cutaneous transpiration, and of the alvine discharge, the average quantity of urine voided in twenty-four hours may be taken to be in the adult from 35 to 60 ounces.

The quantity is diminished in all febrile diseases; in heart affections when compensation is lost, in cases of collapse, and generally in all these conditions in which much fluid is passing out of the blood, such, for example, as profuse diarrhœa or perspiration, the rapid accumulation of serum in the pleuræ or peritoneum, etc. Further, there is scanty urine in the inflammatory form of Bright's disease, whether there be inflammation of the tubules or of the glomeruli. The urinary flow may be completely suppressed in cases where the ureters are occluded by the impaction of calculi, or by the pressure of morbid growths.

The urinary flow is *increased* by the administration of diuretics. It is greatly augmented in cases of diabetes insipi-

dus and mellitus, chiefly owing to the large quantities of water which such patients drink. In the cirrhotic, or contracting form or Bright's disease, the quantity of urine secreted is increased in the later stages, when the heart has become hypertrophied, and the vascular tension increased. On the other hand, in the waxy form (as was first pointed out by Professor Grainger Stewart) polyuria is an early symptom, often occurring even before the presence of albumen can be detected.

Color of the Urine.—The urine owes its color to the quantity of pigment it contains, and to the amount of its concentration—very dilute being pale, very concentrated having a dark brownish-red color. For convenience of comparison, Vogel's standard scale of colors is usually adopted. The various tints are grouped as follows:

Yellow Urines,	Pale yellow, Bright yellow, Yellow.
Red Urines,	Reddish-yellow, Yellowish-red, Red.
Dark Urines,	Brownish-red, Reddish-brown, Brownish-black.

In order to obtain uniform results as to color, the urine should be examined in a glass, the diameter of which is about four inches; and if not absolutely clear, the urine must be filtered before its color is noted.

Very pale urines are met with in healthy persons after copious draughts of water, and further, in cases of diabetes and of anæmia, and after hysterical paroxysms. Highly-colored urines occur in the febrile state, and under other pathological conditions, which will be mentioned more particularly hereafter.

What the pigments of normal urine are is still a matter of doubt. There are, however, two pigments, both of which must be looked upon as pathological, uro-bilin, and uro-erythrin, about which a few words must be said.

Uro-bilin is a reddish pigment, first described by Jaffé,* which is found in considerable quantity in the urine of fever,

* Virchow's *Archiv.*, vols. xlvii. and xlviii.

and sometimes in that of jaundice. In normal urine, when first voided, it does not occur, but it often appears after the urine has been allowed to stand for some time in contact with the air. The chief interest of uro-bilin is derived from its relations with, on the one hand, bilirubin, which, as Maly * has shown, when treated with sodium-amalgam, yields a body which he named hydro-bilirubin, and which is identical in all its characters with uro-bilin; and, on the other hand, with blood pigment, for Hoppe-Seyler has pointed out that when hæmatin in alcoholic solution is treated with tin and hydrochloric acid, uro-bilin is formed. The *detection of uro-bilin* is usually easy, and depends chiefly upon the three following points: (1.) Examined with the spectroscope, most urines which contain uro-bilin show an absorption line between Frauenhofer's lines *b* and F, which is not very well defined, and which shades away towards F. Sometimes, however, the spectrum cannot be made out in the urine itself. In such cases if the urine be shaken up with ether, the etherial solution of the pigment will show the spectrum clearly. (2.) When a small quantity of chloride of zinc is added to an alkaline solution of the pigment, a green fluorescence appears. (3.) The addition of ammonia to the urine itself, or to an acid solution of uro-bilin, changes the reddish color into clear yellow.

Uro-erythrin is a pinkish-red pigment (the purpurin of Bird) which often appears in the urines of fever, and of cirrhosis of the liver, and which attaches itself to precipitates of urates and of uric acid, giving the sediment a brick-dust color. This deposit, however, often occurs in otherwise healthy persons from errors in diet and other slight causes.

Melanin, the black pigment which is found in the urine in cases of melanotic cancer, may at times possess some diagnostic significance.

The administration of certain drugs is followed by alteration in the color of the urine. Thus after the absorption of carbolic acid, the urine becomes of a dark greenish-brown color, due to the presence of an oxidation product of hydrochinon. Rhubarb and senna (chrysophanic acid) color the urine a deep brownish-yellow, which changes to bright red on the addition of an alkali. Logwood imparts a red tinge, and santonin a bright yellow, which changes to orange when ammonia is added.

---

* *Centralbl. f. d. Med. Wissensch*, 1871.

The presence of blood and bile pigments in the urine will be considered hereafter.

Transparency.—Normal urine, when freshly passed, is almost invariably transparent; but when allowed to stand, clouds of mucus form in it, which, at the end of twelve hours, will be found to have sunk to the bottom of the vessel. In highly concentrated urine, and especially in that of the various feverish processes, a dense cloud of urates forms after cooling has taken place, which, as well as the other urinary sediments, will be considered further on in these pages.

Odor.—Freshly-passed normal urine has a faint odor peculiar to itself, which gradually disappears. When it becomes alkaline, an ammoniacal odor develops itself in the urine. When blood or pus becomes added, the urine has a peculiarly offensive odor from its rapid decomposition.

Turpentine, when inhaled or taken internally, imparts an odor of sweet violets to the urine. Copaiba, cubebs, tolu, and asparagus, also communicate a characteristic smell. Finally, in diabetes mellitus, the urine has a faint, sweetish odor, which, if acetonæmia develops itself, comes to resemble that of chloroform.

Specific Gravity.—The specific gravity is usually and most conveniently estimated by means of a urinometer. The instrument is dipped into the urine and allowed to float, the point at which the level of the surface of the urine cuts the graduated stem being read off, and thus the specific gravity is ascertained. One or two precautions, must, however, be taken. The urinometer must be carefully dried before use, as drops of water adhering to the upper part of the stem tend unduly to depress it. It must also float completely clear of the edge of the vessel, and the surface of the urine must be free from air-bubbles, which, if present, can be readily removed by means of filter-paper. As urinometers are graduated for a temperature corresponding to that of an ordinary room, observations must not be made on urines until they have cooled down to that point.

The urinometer scale commences at 1000, the specific gravity of distilled water, and usually goes up to 1050. The average specific gravity of normal urine may be taken to be from 1015 to 1025; but readings both above and be-

low these limits are quite consistent with perfect health. The specific gravity of any urine expresses of course, the quantity of solids which that urine contains in solution. Thus, if we find it in any particular instance to be, let us say, 1025, we know that there are present solids in such quantity as to suffice to raise the weight of a litre of distilled water from 1000 grammes to 1025.

From the specific gravity so obtained, it is possible roughly to calculate the quantity of solids present in the urine. This may be done by means of the very simple formula given by Trapp, which consists in multiplying the two right hand figures by two, the result being the amount of the solids in 1000 parts of the urine.

From what has been said, it is clear that as the specific gravity of the urine depends upon the proportion of solids to fluid, it will be affected by changes in the quantity of either. Thus, after copious imbibition of water, the urine of healthy persons may have a specific gravity as low as 1002; and, on the contrary, after profuse perspiration, it may rise to 1040. We must thus take into account the *quantity* of the urine passed in twenty-four hours before we allow ourselves to judge what importance is to be attached to the specific gravity. When the quantity is large, we find, if the urine be normal, a low specific gravity; whereas, when the flow is scanty, the specific gravity is high. If, however, we meet with a urine which, while large in quantity, possesses a high specific gravity, or one which, while small in amount, is low in gravity, then the fact may in each case be noted as distinctly pathological.

Pathological urines may be classified as follows:

High Specific Gravity is found after copious perspiration, vomiting, or purging, owing to the consequent concentration of the urine. At the commencement of all acute feverish diseases, the specific gravity of the urine is high, running up even to 1035, and this owing, in part, to diminished watery excretion, but also, in great measure, to the inincreased elimination of urea, sulphates, and phosphates which then takes place. Much more marked and important, however, is the increase of specific gravity met with in the urine of diabetes mellitus. In this disease we find large quantities of urine being passed, the specific gravity of which varies from 1030 to 1060, its height being due to the presence of grape sugar.

Low Specific Gravity, when not due to the great dilution

of the urine, is commonly the result, either of some disturbance the secreting apparatus of the kidney (Bright's disease, circulatory disease, etc.), or of general interference with nutrition (anæmia, cachexia, etc.), in both cases arising directly from the defective elimination of the urinary salts, particularly urea and its compounds.

Reaction.—The reaction of the urine may be tested by means of blue and red litmus paper. Normal urine is acid when fresh, very rarely neutral or alkaline, the acidity being due to the presence of free acids—such as lactic, oxalic, hippuric, and acetic, and acid salts. After a meal, the urine loses in acidity, sometimes becoming neutral or even alkaline; but it very rapidly regains its former character. The effect of both warm and cold baths is to render the urine alkaline; and the same result is produced much more powerfully by the action of such alkaline medicines as the bicarbonates and acetates of potassium and sodium. The effect of the administration of acids in cases of alkaline urine is not so powerful; but by means of carbonic and benzoic acids, acidity may be produced. Alkalinity of the freshly-passed urine may either be due to the presence of a fixed alkali* (in which case it probably results from some debilitating influence acting upon the system generally), or to the presence of ammonia. The latter form, which is by far the most common, points to some local disease in the bladder or urethra. Ammoniacal urine is frequently met with in cases of long-standing urethral stricture, chronic cystitis, spinal affections, etc. Highly acid urine, on the other hand, is met with in acute febrile diseases, and especially in acute rheumatism.

Normal urine undergoes, when kept too long, fermentative changes, which, as they are liable to cause mistakes, must be carefully noted.

1. *Acid Fermentation.*—If the urine be allowed to stand exposed to the air, in a cool place, it will be found that its reaction increases in acidity steadily from day to day, and may continue to do so for as long as ten days. This fermentation is due to the presence of a peculiar organism,

* If the alkalinity be due to the presence of ammonia, the red litmus paper, which has been turned to blue by dipping in the urine, will regain its red tint after drying; but if the alkali be a fixed one, the blue tint will be permanent.

resembling yeast, but smaller, and is accompanied by the precipitation of a yellowish-brown sediment consisting of uric acid and urates, and frequently of oxalate of lime, along with clouds of mucus. The acidity is probably due to the formation of lactic and acetic acids.

2. *Alkaline Fermentation.*—After the acid reaction has fully developed itself, it gradually disappears, and the urine becomes alkaline. This change does not usually set in, when the urine is kept cool, before eight or ten days have passed; but if there be much pus or mucus present, the alkaline reaction may be detected much sooner; and if any admixture of old, decomposed urine be allowed to take place (as from the glass not having been thoroughly cleaned), it may come on in a few hours. The urine now becomes lighter in color, opaque, and ammoniacal in odor, the urea having become changed into carbonate of ammonia. A white sediment separates, consisting of urate of ammonia, triple phosphate, amorphous phosphates, and carbonate of lime.

CHAPTER XXIII.

URINARY SYSTEM.—(*continued*).

NORMAL CONSTITUENTS OF URINE.

THE normal constituents of the urine may be divided into two classes—organic and inorganic. Of these the following, which are the most important, will be here considered:

Organic Substances.—1. Urea; 2. Uric Acid; 3. Creatinin; 4. Indican.

Inorganic Substances.—1. Chlorides; 2. Sulphates; 3. Phosphates.

Urea is by far the most important constituent of normal urine, and it is to be regarded as the chief product of the decomposition of albumen, and the last product of the regressive metamorphosis of the nitrogenous tissues of the body. The quantitative estimation of urea is therefore of much importance. It may be carried out in either of two ways.

(1.) *Estimation of Urea by means of Nitrate of Mercury.*—

This method, which was first introduced by Liebig, depends the property which urea possesses of forming an insoluble compound with nitrate of mercury. When a dilute solution of the mercury salt is added to a solution of urea, this precipitate forms so long as any urea remains unaltered. If, however, more nitrate of mercury be added when no more urea is present, a drop of the mixture when added to a solution of carbonate of soda gives a yellow precipitate of the hydrated oxide of mercury. In this way can be determined the exact point at which all the urea has been decomposed. Before this method can be applied, however, the phosphates and sulphates which the urine contains must be precipitated by means of a solution of baryta. For the volumetric analysis of urea we therefore require three solutions.

1. *A solution of nitrate of mercury*, of which 1 cub. centimetre corresponds to 0.01 gramme of urea. The method of preparation will be described in Appendix B.
2. *A solution of baryta*, prepared by mixing one volume of a cold saturated solution of nitrate of baryta with two volumes of cold saturated baryta water.
3. *A solution of carbonate of soda* of about twenty grains to the ounce.

The urine must first be freed from phosphates and sulphates, and for this purpose 40 c.c. are measured off by means of a pipette, and 20 c.c. of the baryta mixture added. After filtration, 15 c.c. of the filtrate (corresponding to 10 c.c. of the original urine) are measured off into a small beaker, which is placed under a graduated burette containing the standard solution of nitrate of mercury. From this burette small quantities of the solution are successively added to the urine, the mixture being all the time carefully stirred by means of a glass rod, and the additions are to be cautiously continued so long as a distinct precipitation follows each drop. When, however, the formation of the insoluble compound appears to be getting less distinct, and the analysis consequently approaches completion, a drop of the mixture must be removed on the stirring rod and added to a small quantity of the solution of carbonate of soda, which has been placed on a porcelain plate, or better, in a watch-glass with a black background. Should a white precipitate form, the addition of mercury must be continued, but as soon as a yellow color appears when a drop

is added to the soda solution, the analysis is at an end. The quantity of the nitrate of mercury solution which has been used is now to be read off, and a calculation made, remembering that every cubic centimetre corresponds to 0.01 gramme of urea. Corrections are, however, under certain circumstances, necessary. If, in the course of the analysis, it be found that more than 30 c.c. of the mercury solution are being used for 15 c.c. of the urine mixture, we must add to the urine, before applying the carbonate of soda test, half the number of cubic centimetres of water as we have used of mercury solution above 30. Thus, if 48 c.c. of the standard solution have been dropped in, we should have to add 9 c.c. of water before we transferred a drop to the carbonate of soda solution. On the other hand, if less than 30 c.c. of the mercury solution have sufficed to precipitate all the urea, we must subtract from the total sum of the mercury solution used 0.1 c.c. for every 5 c.c. less than 30. For example, if only 20 c.c. of the standard solution have been used, we must substract 0.2, leaving 19.8 as the sum from which we have to calculate. If great accuracy be required, the chlorides should be previously removed by precipitation with nitrate of silver. The modifications in this method which have been recently suggested by Pflüger* are too complicated to be described here.

(2.) *The Estimation of Urea by means of Hypobromite of Soda.*—In principle this method was first described by Davy. It depends upon the fact that urea, when treated with hypobromite of soda, breaks up into nitrogen, water, and carbonic acid, the last of which is absorbed in the alkaline solution, while the nitrogen comes off as free gas. Of the many forms of apparatus which have been described and are used for this analysis, perhaps the most simple is that of Dr. Graham Steele.† It consists of an ordinary burette inverted in a tall glass cylinder containing water, and connected with a small conical glass vessel containing a short test-tube. After removing the test-tube, the conical vessel is filled to the depth of about an inch with the solution of hypobromite of soda (the method of preparing which will be presently described), and into this the test-tube is carefully slipped, after 5 c.c. of the urine to be tested have been placed in it. The cork of the conical ves-

---

* *Arch. f. d. ges. Physiol.*, xxi. and xxiii.

† *Edin. Med. Journal*, 1874.

sel is then replaced, and the vessel dipped into water. The burette, which is now in communication with the conical vessel, is next raised or lowered as may be required, until the level of the water inside and outside is the same, and this point is read off. The conical vessel is now tilted over so as to allow the urine in the test-tube to flow out and to become mixed with the hypobromite solution. This mixture is followed by a rapid giving off of gas, and after all effervescence has ceased, and the nitrogen which has collected in the burette has had time to cool down to the temperature of the room, the burette is again moved so as to bring the water-level inside to the same height as that outside, and this point read off. The difference of the two readings gives the quantity of nitrogen which has been given off. Since we know that at the ordinary temperature of a room, 0.1 gramme of urea gives off 37.5 c.c. of nitrogen, the calculation is simple. This method, from the ease with which it can be carried out, is very convenient, but it is not extremely accurate, for not only urea, but also uric acid and creatinin, give off nitrogen when treated with hypobromite of soda. The error is, however, small. The preparation of the solution of hypobromite of soda is made as follows: 100 grammes of caustic soda are dissolved in water, and the solution diluted to 1250 c.c. To this 25 c.c. of bromine are to be added, and the whole shaken vigorously. This solution must be kept in a stoppered bottle and in the dark. It decomposes rapidly, and can only be used when freshly prepared.

The quantity of urea excreted in twenty-four hours in healthy men averages from 300 to 500 grains. It is increased after exertion, and after a full meal of animal food. In almost all diseases attended with elevation of temperature, the urea elimination is increased. Thus in typhus, pneumonia, pleurisy, and acute rheumatism, the amount of urea excreted is usually much above normal. In diabetes mellitus, I have seen the daily quantity to be as high as 1800 grains. This is no doubt in part due to the large quantity of animal food consumed in this disease. On the other hand, the urea is diminished in almost all affections of the kidney, owing to defective eliminating power of that organ. Particularly is this the case with regard to acute inflammatory Bright's disease, and to the cirrhotic or contracting form, especially when in its later stages a degree of inflammatory action becomes superadded.

The urea excretion is affected by the administration of drugs, but much uncertainty exists as to the action of many of these. Phosphorus undoubtedly increases the elimination of urea, as do also most, if not all, diuretics. Morphia, quinine, and iodide of potassium, on the contrary, tend to diminish its quantity.

Uric Acid exists in normal urine in combination with potassium, sodium, ammonium, calcium, or magnesium; and, as all these salts of uric acid are very much more soluable in hot than in cold urine, they tend to separate out as the urine cools. The cloud of urates which thus so often appears soon after the urine is passed may be readily recognized by warming a small quantity of the urine containing the sediment in a test-tube, when it rapidly becomes clear. Uric acid is readily separated from urine by adding hydrochloric adid. It then deposits itself in crystalline form, the character of which will be described when we come to speak of urinary sediments.

Detection of Uric Acid.—It is often important to be able to detect the presence of uric acid in concretions and sediments. Very frequently this may be done by means of the microscope, but this is not always possible. Uric acid can however always be detected by means of the *murexid test*, which is applied as follows: A small quantity of the sediment is dissolved in a porcelain dish with a few drops of nitric acid, and the solution so obtained is evaporated. To the reddish residue one or two drops of dilute ammonia are added, when the beautiful reddish purple color of murexid develops itself, which, on the further addition of a few drops of caustic potash, passes into bluish purple.

The quantitative estimation of uric acid is difficult to carry out. A large quantity of urine (100-200 c.c.) is taken, and to it is added 5 c.c. of pure hydrochloric acid. The mixture is allowed to stand for forty-eight hours, and the precipitate of uric acid is then collected on a filter, washed with a little cold water, and weighed. Salkowski has however recently pointed out* that by no means all the uric acid which the urine contains is thus removed by precipitation, and that the proportion which remains varies much in different urines. He recommends that the filtrate should be further treated with ammonia and magnesia, and after

* Virchow's *Archiv.*, vol. lii.

the precipitate of phosphates has been removed by rapid filtration, that the filtrate should be further treated with an ammoniacal silver solution. The precipitate which is thus formed, after careful washing, is then decomposed with sulphuretted hydrogen, the filtrate acidulated with hydrochloric acid, and the uric acid which then separates out collected and weighed.

The average quantity of uric acid excreted in twenty-four hours is about 0.5 to 1 gramme. It rises and falls simultaneously with urea, bearing the relation to that substance of 1 to 50 or 60 in the healthy state. It is much increased by an animal diet. In most feverish conditions the excretion of uric acid corresponds with that of urea, but when the respiration is interfered with, Bartels states that it is increased. During an attack of gout the excretion is diminished, but after the paroxysm is over, it undergoes some increase. It is increased in many hepatic affections, in leucocythæmia, in acute rheumatism, and sometimes in cases of simple indigestion. The appearance of a sediment of uric acid is only to be looked upon as pathological when it occurs either before the urine cools or immediately thereafter.

Creatinin is a normal constituent of urine, and is present in somewhat larger quantity than uric acid. Its presence is readily detected by adding to a small quantity of urine a few drops of a very dilute solution of nitro-prusside of sodium, when, on the further addition of dilute caustic soda a beautiful ruby red color develops itself, which soon passes into deep straw yellow.

Quantitative Analysis of Creatinin.—300 c.c. of urine are taken, rendered alkaline by the addition of milk of lime, and then decomposed with chloride of lime until no more precipitation takes places. The filtrate is rapidly evaporated to the consistence of syrup, and mixed with 50 c.c. of alcohol (95 per cent.). The mixture is allowed to stand until all the chloride of sodium has separated out, is then filtered, and the filtrate evaporated down and treated with an alcoholic solution of chloride of zinc. After standing for three days, the zinc-creatinin chloride will have become fully separated, and may then be collected on a filter and weighed.

The quantity of creatinin excreted, which is normally about one gramme *per diem*, is increased in typhus and

pneumonia, and diminished in anæmia, chlorosis, and tuberculosis.

Indican.—It is not uncommon to encounter dark-yellow urines, in which, on the addition of nitric acid, there becomes developed the dark violet color of indigo. The original pigment which is thus decomposed was first studied by Schunck,* who took it to be identical with the indican of plants. Later investigations have however shown that the two substances are not quite identical, and that the indican found in urine is to be looked upon as potassium-indoxyl-sulphate. It is identical with the uroxanthin of Heller. Indican, when treated with a mineral acid, yields indigo, and this decomposition often takes place in the urine spontaneously after decomposition has set in, the indigo appearing on the edge of the glass and on the surface of the urine as a glistening dark-blue film. Indican is a derivative from indol, which is a result of the changes which albumen undergoes in the intestines, and Jaffé was the first to point out† what was subsequently confirmed by Baumann and others, that when indol was injected subcutaneously in animals indican appeared in quantity in the urine.

Detection of Indican.—The original process of Jaffé as modified by Senator‡ is as follows: In a large test-tube are placed 10-15 c.c. of the urine, and to this is added an equal quantity of fuming hydrochloric acid, and then a concentrated solution of chloride of lime drop by drop, until the blue color is fully developed. The mixture is now to be shaken up with chloroform, after which the chloroform will sink to the bottom, more or less deeply tinged with indigo. By this means a rough idea of the quantity of indican present may also be arrived at.

The method of estimating accurately the quantity of indican as given by Jaffé,§ is very complicated, and for its details the original must be consulted.

The quantity of indican contained in human urine averages 6.6 mgrm. in 1000 c.c. In disease the indican excretion has been frequently investigated, most fully perhaps by

---

* *Proceedings of the Royal Society*, 1857.
† *Centralbl. f. d. med., Wiss.* 1872.
‡ Ibid. 1877, p 357.
§ Pflüger's *Archiv.*, iii.

Hennige.* The most important clinical point is that indican appears in large quantity in the urine when there is obstruction of the small intestine, while, when the obstruction is seated in the large intestine, no such augmentation takes place. In cases of ileus, therefore, when the exact seat of the disease is doubtful, the estimation of the indican in the urine may have considerable diagnostic value.

Passing now to the consideration of the inorganic substances which are found in normal urine, we come first to the

Chlorides.—The chlorine which is contained in urine exists in combination with potassium, sodium, ammonium, magnesium, or calcium. The presence of these chlorides may be detected by adding to a small quantity of urine in a test-tube a few drops of nitric acid, and then a small quantity of a solution of nitrate of silver. A white flocculent precipitate at once falls, consisting mainly of chloride of silver, but also containing combinations of silver with uric acid, creatinin, xanthin, and urinary pigments.

Estimation of the Chlorides.—Mohr's method depends upon the fact that when to a neutral urine containing chloride and phosphate of sodium and a neutral salt of chromic acid, a solution of nitrate of silver is added, there first occurs a precipitation of chloride of silver; and when the point is reached when all the chlorine contained in the chloride of sodium is so precipitated, there then begins the precipitation of the red chromate of silver. For this analysis we therefore require—

1. *A solution of nitrate of silver*, of which 1 c.c. corresponds to 10 milligrammes of chloride of sodium or to 6.065 milligrammes of chlorine.
2. *A cold saturated solution of neutral chromate of potassium.*
3. *Pure nitrate of potassium.*
4. *Pure carbonate of lime.*

Since the presence of urinary pigments prevents the accuracy of this method, the silver combining with them, they must first be got rid of in the following manner:

10 c.c. of urine are mixed with 2 grammes of nitrate of potassium in a platinum capsule, evaporated to dryness

* *Deutsches Archiv. f. kl. med.*, xxiii., p. 271.

and finally heated in a naked flame until the carbon is completely oxidized. The residue is then dissolved in water in a beaker, acidulated with a dilute solution of pure nitric acid, and then neutralized with a little carbonate of lime. To the fluid so obtained, which need not be filtered, four or five drops of the chromate solution are added, and then the silver solution is gradually dropped into it from a burette, the mixture being constantly stirred. Reddish spots appear where the solution falls, but they disappear on stirring, so long as any chloride of sodium is present. So soon, however, as the whole of that salt is decomposed, the next drop of the silver solution gives rise to a permanent red which marks the conclusion of the operation. The amount of the silver solution which has been used is now read off, and as we know that each cubic centimetre corresponds to 6.065 milligrammes of chlorine, the calculation is easy.

The average quantity of chlorine excreted in the urine in twenty-four hours may be taken to be in the healthy state about 10 or 12 grammes. It is increased by the consumption of a greater quantity of common salt, and by the copious drinking of water.

In disease the most important change which occurs in the elimination of the chlorides is the remarkable diminution met with in acute feverish conditions, particularly in pneumonia. As the result of a very interesting series of observations, Röhmann * comes to the conclusion that this diminution is due, not to a decrease in the amount of chlorine taken along with the food, but to a change in the relation of the albumen of the blood to the chloride of sodium in the plasma.

Sulphates.—The sulphates which are found in the urine are derived from the breaking up of albumen, either that of the tissues or that which is contained in the food. Sulphuric acid exists in the urine in two forms—first, in combination with the alkalies; and, second, as Baumann has shown, in the form of aromatic ether-sulphuric acid—chiefly phenyl-sulphuric acid and indoxyl-sulphuric acid. Both of these aromatic bodies when heated with hydrochloric acid break up into phenol or indigo and sulphuric acid. Acetic acid does not cause this decomposition.

---

* *Zeitschrift. f. kl. med.*, vol. i., p. 513.

Detection of the Sulphates.—Acidulate strongly with acetic acid, and on the addition of chloride of barium a white precipitate of sulphate of barium will fall, representing the sulphuric acid which was combined with the alkalies. If now the mixture be filtered and heated with hydrochloric acid, a further precipitate of sulphate of barium will fall, representing in this case the ether-sulphuric acid.

Estimation of the Sulphates.—If these two precipitates which have just been mentioned be weighed, the total amount of the sulphates, as well as that of each form, may be calculated. Fürbinger * has pointed out an easy manner of obtaining results which are approximately accurate, and quite sufficiently so for comparative observations, which is to wash the precipitates in question into a very narrow graduated cylindrical vessel, and allow them to settle down. After some hours their upper level may be read off, and if the physician possesses the result of only one weighing of such a precipitate, he can always translate into *weight* the height of his precipitates. For the purpose of such an analysis, however, at least 300 c.c. of urine must be taken.

The normal quantity of sulphuric acid which is excreted in the urine in twenty-four hours is about two grammes. It is increased and diminished according as more or less albumen is broken up, and therefore it corresponds with the quantity of urea and uric acid, both in health and in disease.

Sulphur is also excreted in the urine in small quantity in the form of sulphocyanic acid, of taurin,† and occasionally of cystin. Sulphuretted-hydrogen is also sometimes met with in the urine in disease.

Phosphates.—In normal acid urine phosphoric acid is met with in the form of the phosphates of the alkalies, sodium and potassium, and of calcium and magnesium. It may also appear in the form of glycerin-phosphoric acid and lecithin. It is derived in part from the food, and in part from the breaking down of tissues of the body which contain phosphorus, principally the osseous and the nervous structures.

When the urine loses its carbonic acid, as it does when heated, the earthy phosphates separate out as a white floc-

* Virchow's *Archiv.*, vol. lxxiii.
† Salkowski-Virchow's *Archiv.*, vol. lviii.

culent precipitate, which becomes redissolved on the addition of acid. The addition of ammonia to urine causes an amorphous precipitate of phosphate of lime, while the phosphate of magnesium unites with the ammonia to form ammonio-magnesian phosphate (triple-phosphate), which appears in a crystalline form. The microscopic appearance of all the various forms of phosphate will be described when we come to speak of urinary sediments.

Estimation of Phosphoric Acid.—The principle of Neubauer's method is the following: When a hot solution of the phosphates in question is acidulated with acetic acid, it gives, with a solution of acetate of uranium, a precipitate of uranium phosphate. The point at which this reaction ends is, from the nature of the precipitate, difficult to determine, and it is consequently necessary to test the mixture from time to time with a solution of ferro-cyanide of potassium, which gives, when there is present the slightest excess of the uranium solution, a dark reddish-brown coloration. The solutions required are:

1. A solution of uranic oxide of which 1 c.c. is equivalent to 0.005 gramme of phosphoric acid, P_2O_5.
2. A solution of acetate of soda prepared by dissolving 100 grammes of that salt in 900 c.c. of water, and adding 100 c.c. of concentrated acetic acid.
3. A solution of ferro-cyanide of potassium not too concentrated.

To 50 c.c. of the urine are added 5 c.c. of soda solution, and the mixture is placed in a beaker glass, and warmed in a sand bath. From a burette the uranium solution is gradually added to the urine, until no further precipitation appears to take place. A drop is now removed, placed on a porcelain slab and mixed with a drop of the solution of ferro-cyanide of potassium. If there be any excess of uranium—*i.e.*, if the analysis be at an end—a reddish-brown precipitate will appear where the drops come in contact. If this reaction does not take place, more uranium solution must be added to the urine. Each cubic centimetre of the uranium solution used corresponds to 0.005 gramme of phosphoric acid, so that the calculation is easy.

If it is wished to estimate separately the earthy phosphates, these must be precipitated by the addition of ammonia, the precipitate carefully separated by filtration,

dissolved in water with the addition of a little acetic acid, and the solution treated in the manner just described.

The average quantity of phosphoric acid which is excreted in the urine in twenty-four hours is in the adult about three grammes, two thirds of which may be taken to consist of the phosphates of the alkalies, and one third of earthy phosphates. The quantity depends to a large extent upon the food—animal diet giving rise to more excretion than vegetable—and upon the condition of the alvine secretion, the earthy phosphates in particular being much increased in quantity when there is much constipation. Tissue change also influences the phosphatic elimination to a large extent, chiefly that which takes place in the nervous structures.

In the feverish state the phosphates are at first diminished, but when convalescence sets in their amount in the urine is increased to a point above normal. In chronic nervous diseases the phosphates are usually present to an excessive amount in the urine, and in ostiomalacia the earthy phosphates are increased to such a degree that they may be found to be in excess of the phosphates of the alkalies. In meningitis also the phosphates have been found to be increased, and this fact is often useful in the differential diagnosis of that complaint.

CHAPTER XXIV.

URINARY SYSTEM—(*continued*).

ABNORMAL CONSTITUENTS OF URINE.

THE substances which are commonly grouped under this heading are some of them present in normal urine, as, for example, sugar and oxalic acid ; but their quantity is then so small as to elude detection by the ordinary methods of analysis, and it is only under pathological conditions that they appear in sufficient quantity to require notice. Other members of this group are, however, never present in normal urine.

Albumen.—The presence of albumen in the urine is one of the most important diagnostic indications which the

physician can encounter. The special circumstances under which albuminuria occurs will be detailed presently. In the meantime we have to consider the methods by means of which its presence may be detected, and its quantity estimated.

The chief albuminous substances which appear in the urine are—

1. Serum-albumen.
2. Serum-globulin (paraglobulin).
3. Propepton.
4. Fibrin.

The first two of these substances are both detected by the ordinary tests for albumen. Their separation will be subsequently considered.

Detection of Albumen (serum-albumen and serum-globulin).—Before testing for albumen, the urine in question must, if not already clear, be rendered so by careful filtration. Of the many methods employed, the following are the most important :

(1.) *Boiling Test.*—If a small quantity of urine be placed in a test-tube, and heated in the flame of a spirit-lamp or Bunsen burner, it will be found that when the temperature has risen to near the boiling point the albumen, if present, separates out as a white cloud, which, on standing, collects at the bottom of the tube in fine flakes. If the urine contain much earthy phosphates, these are apt to separate when the tube is heated, and the cloud so formed may be mistaken for albumen. It is, however, dissolved on the addition of a few drops of acetic acid. If the urine be alkaline to begin with, the albumen may not be separated out on boiling. It is therefore necessary to acidulate with a few drops of acetic acid ; but inasmuch as there is some risk of adding too much of this acid, and so preventing the albumen reaction from taking place, it is best to proceed in all cases as follows :

5-10 c.c. of urine are placed in a test-tube acidulated with acetic acid, and 1-6 of its volume of a concentrated solution of sulphate of magnesia added. If albumen be present, there will now appear on heating a more or less distinct cloudiness. This test is absolutely trustworthy, and in point of delicacy is probably surpassed by none.

(2.) *Nitric Acid Test.*—A small conical glass is taken and filled about one third full of urine. Down its side, while

it is held inclined, are poured slowly a few drops of strong nitric acid, in such a way that when the glass is again held in an upright position, the acid forms a distinct layer at the bottom. If albumen be present in the urine, a cloud will form at the line of junction of the two fluids.

If the urine contain a large quantity of neutral urate of sodium or ammonium, the addition of nitric acid may cause the separation of the acid urates in the form of a cloud. This cloud lies near the upper surface of the urine, and is therefore not readily mistaken for albumen; but in cases of doubt it is only necessary to warm the glass, and so cause solution of the cloud, or to dilute the urine previously with twice or thrice its volume of water, after which no such cloud will form.

(3.) *The Ferrocyanide Test.*—To the urine contained in a test-tube a drop or two of acetic acid is to be added, and then a small quantity of a solution of ferrocyanide of potassium. If albumen be present, a white flocculent precipitate will separate out in the cold.

Other tests, such as those in which carbolic, tannic, and metaphosphoric acids are employed, are wholly unnecessary.

Estimation of Albumen.—It is often of great importance to the physician to know the quantity of albumen which is being excreted in the urine from day to day. Unfortunately there is no very ready method of performing such an analysis. The most accurate is that of Berzelius. The urine is carefully filtered—10-15 c.c. of the filtrate placed in a porcelain dish, carefully acidulated with acetic acid and evaporated to dryness on a water-bath. The remainder is extracted first with hot water, and then with alcohol, placed upon a weighed filter, dried at 100°C., and finally weighed. From the result so obtained must be subtracted the quantity of earthy phosphates and coloring matter which the residue contains, and this is done by burning the filter and the coagulum in a platinum capsule, and deducting the weight of the ash so obtained.

The quantity of albumen present in urine may likwise be estimated by means of Laurent's polarimeter. The urine must be rendered very clear by means of filtration, or, if this fail, by the addition of a little milk of lime and subsequent filtration. If the tube 1 decimeter long be used, then each degree of polarization to the left corresponds to

1 gramme of albumen in 100 c.c. of urine. This method of estimating albumen is, however, unfortunately not very accurate.

Serum-globulin (Paraglobulin) may be readily detected in urine by means of Hammarsten's method.* If the urine be saturated with magnesium sulphate, which is to be added in the form of a fine powder, the globulin will separate out as a white flocculent precipitate. According to Estelle,† the best method for estimating separately the serum-albumen and the serum-globulin is as follows: He takes a small quantity of urine and adds to it sulphate of magnesia until no more will dissolve. The mixture, after having been shaken for ten minutes, is poured upon a weighed filter, the precipitate washed with hot water, and finally with distilled water, until the sulphate of magnesiun is completely removed, as indicated by the fact that chloride of barium when added to the washings gives no longer a precipitate. The filter containing the precipitate is then dried and weighed. In this way the quantity of serum-globulin is obtained. The serum-albumen which was contained in the original quantity of urine is now contained in the first filtrate, and its amount can be estimated by coagulating with acetic acid, and boiling, filtering, and weighing in the manner already described. The clinical bearings of serum-globulin will be presently referred to.

Propepton.—The peculiar albuminous substance, which Bence Jones first described‡ as occurring in the urine of a patient suffering from mollities ossium, is in all probability identical with Schmidt-Mühlheim's propepton and with Kühne's hemi-albumose. It is an intermediate product of the digestion of pepsin or trypsin before pepton is formed. Virchow found§ it to be present in the medullary substance of the bones in cases of mollities ossium, Lassar detected it‖ in the urine of petroleum poisoning, and Neale¶ in a case of hæmoglobinuria. Its chief peculiarity is that though precipitated by nitric acid in the cold it becomes dissolved on heating, and again separates out when the mixture is allowed to cool.

---

* Pflüger's *Archiv.*, vols. xvii. and xxii.
† *Revue Mensuelle*, 1880. p. 704.
‡ *Phil Trans.*, 1848.
§ Virchow's *Archiv.*, iv.
‖ Ibid., lxxvii.
¶ *Lancet*, 1879.

Detection.—The urine must first of all be freed from serum-albumen and serum-globulin. If the urine be acidulated with acetic acid, saturated with sulphate of magnesia, heated to boiling and then filtered hot, these two substances will remain on the filter while the propepton will pass through in solution in the hot filtrate, and will separate out as cooling takes place.

Fibrin, when present in the urine, is usually in the form of flakes, and is due to inflammatory action in the kidneys and urinary passages. It is probable that the casts of the renal tubules, of which we shall presently come to speak, are chiefly composed of fibrin. The spontaneous coagulation of chylous urine is due to the presence of fibrin.

The transitory occurrence of albumen in the urine has been frequently observed in persons who are apparently in good health. Such cases have been described by Ulzmann.* Furbringer,† Bamberger,‡ Runeberg,§ and others. Apart from such intermittent albuminuria, the chief conditions which give rise to albuminuria of a more or less permanent character may be grouped as follows:

(1.) In most febrile conditions albumen may appear in the urine, but usually only in small quantity. When the amount is considerable, it points to the occurrence of Bright's disease as a complication.

(2.) In such affections of the heart or lungs which lead to circulatory changes in the kidneys, in particular to venous engorgement, albumen appears in the urine; but here, also, the quantity is not very great.

(3.) In Bright's disease. In all the different forms of this affection, albumen appears in the urine. Its amount is greatest in the inflammatory form (in severe cases the urine may even become solid on heating), and whenever we find a large quantity of albumen, the presence of this form of Bright's disease must be suspected. In the cirrhotic and waxy forms, when these are uncomplicated with inflammation, the albumen is usually only present in very small amount; indeed, albuminuria often does not show itself until some considerable time after the commencement of the morbid process in both of these disorders, and when it does

---

* *Wiener med. Presse*, 1870.
† *Deutsch. Archiv. für kl. med.*, vol. xxvii.
‡ *Wiener med. Wochenschr.*, 1881.
§ *Deutsch. Archiv. für kl. Med.*, vol. xxvi.

set in it is often subject to distinct remissions, appearing one day and disappearing the next. It is of great importance to distinguish the albuminuria of Bright's disease from that which occurs in other disorders, and particularly from that of heart disease; and while a consideration of the whole circumstances of the case will generally lead to a correct diagnosis, the most important point is the presence of renal tube-casts and epithelium, which are usually more or less abundant in Bright's disease.

(4.) Various nervous disorders are accompanied with albuminuria. It occurs, for example, after an epileptic fit, and Warburton Begbie was the first to point out* that albumen was frequently to be found in the urine in cases of exophthalmic goitre.

Albuminuria also occurs in cases of lead poisoning, and sometimes in pregnancy.

In regard to the occurrence of serum-globulin little of diagnostic importance is known. Senator states that while it is almost always present in albuminous urine, it is most abundant in the waxy form of Bright's disease. In the article to which I have already referred, Estelle states that it is invariably to be found in albuminuria; that it is often present in greater quantity than the serum-albumen; and that sometimes it exists alone.

The albumen found in the urine may in certain cases be derived from blood, pus, or spermatic fluid with which the urine has become mixed.

Pepton.—Closely allied to these albuminous substances stands pepton, which Gerhardt was the first to describe as occurring in urine. The various peptons, which are the products of the digestive action of pepsin and the pancreatic juice upon albumen, are not precipitated by acetic acid and ferrocyanide of potassium (as all other albuminous substances are, but are thrown down by tannic acid, phosphor-wolframic acid, and certain other reagents. Peptons further give the biuret reaction (purple-red) with sulphate of copper and caustic soda, and also a red color with Millon's reagent.

To detect pepton in albuminous urine the albumen must be completely separated by means of acetic acid, boiling and filtration, and then by the addition of hydrated oxide

* *Edin. Med. Journal*, 1874.

of lead, the lead being subsequently removed by means of sulphuretted hydrogen. This process must be repeated until no trace of albumen can be detected by the ferrocyanide test. The pepton in the filtrate is then precipitated by means of a solution of tannin, the precipitate collected and washed, the tannin removed by means of the hydrate of baryta, and the baryta by means of dilute sulphuric acid. Finally, the clear filtrate which is thus obtained is tested with Millon's reagent, when a red color will show the presence of pepton.

The subject of peptonuria has been chiefly investigated by Maixner* and Hofmeister.† According to the former observer, pepton appears in the urine very frequently in those diseases in which the formation and collection of pus play a prominent part, such as purulent effusions into the pleural and peritoneal cavities, abscesses in various situations, pyonephrosis, bronchorrhœa, and phthisis when cavities have formed. Peptonuria also occurs, according to the same author, in the stage of resolution of croupous pneumonia, in phosphorus poisoning (confirmed by the observations of Schultzen and Riess), in typhoid fever, and in carcinoma of the stomach.

The appearance of pepton in the urine is to be explained on the supposition that when it passes into the blood it does not undergo the usual change, and so reaching the kidneys as pepton, it is excreted as such. Plosz and Gyergyai‡ have shown that when peptones are injected into the blood-stream, provided that they are not thrown into the portal circulation, they appear unchanged in the urine.

Mucus.—A small quantity of mucus is present in normal urine; but in such affections as catarrh of the bladder or urethra, it may be much increased. It is sometimes of importance to be able to distinguish mucus from pus in the urine. This is readily done by filtration, when, if pus be present, the filtrate will give the reactions of albumen; but if it contain mucus it will give that which is characteristic of mucin—*i. e.*, when acidulated with acetic acid a precipitate of mucin separates out in the cold.

Sugar.—Even in normal urine a small quantity of grape sugar is present; but its quantity is so minute as not to

---

* *Prager Vierteljahrschrift*, cxliii. (1879), p. 78.
† *Zeitschrift für phys. Chemie.*, iv. (1880), p. 253.
‡ Pflüger's *Archiv.*, vol. x.

give evidence of its presence with the ordinary tests which are about to be described. When, therefore, sugar is detected by their means, it is present in abnormal amount, and constitutes the pathological condition termed glycosuria.

Qualitative Tests for Sugar.

The qualitative tests for urine containing sugar depend upon the coloration caused by boiling with caustic potash, upon the power grape sugar possesses of reducing hydrated oxide of copper, and upon the evolution of carbonic acid when fermentation is set up by the addition of yeast. In all cases albumen, if present, should be got rid of by coagulation and filtration before these tests are applied.

1. *The Caustic Potash Test* (Moore's).—The urine is mixed in a test-tube with an equal quantity of liquor potassæ, and the upper part of the mixed fluid heated to boiling in the flame of a spirit-lamp. If sugar is present the heated portion will assume a dark brown color. Almost all urines, it must be remembered, darken slightly when thus treated; but the change is very marked when sugar is present. This test is not very delicate, but is readily performed, and is useful as a preliminary.

2. *Trommer's Test.*—To a small quantity of urine in a test-tube, $\frac{1}{3}$ of its volume of liquor potassæ is added, and then a drop or two of a solution of sulphate of copper. The precipitate which falls will redissolve (the more readily if sugar be present), and more of the copper solution must be added until a small quantity of the hydrated oxide remains as a precipitate. On boiling this mixture a yellow color will show itself if sugar be present, and will pass into a reddish-yellow granular precipitate of the suboxide of copper.

3. *Test with Fehling's Solution.*—Of all the tests for sugar this is by far the most delicate and satisfactory. The method of preparing Fehling's solution will be described further on. A small quantity of that solution is placed in a test-tube heated to boiling, and then a drop or two of urine added. If sugar be present, reduction of the copper in Fehling's solution will at once take place, giving rise to a red precipitate. Fehling's solution is liable to undergo decomposition when kept for some time, and it will then of itself become reduced on boiling. If, however, it be always boiled previous to the addition of the urine, no error can

take place, for if the solution remain clear on boiling, it is in a fit state for use.

4. *Fermentation Test.*—Under the influence of yeast, grape sugar breaks up into alcohol and carbonic acid, and this evolution of carbonic acid has been made the basis of another qualitative test for the presence of sugar. It is most readily performed by taking two test-tubes or narrow phials, one filled with water and the other with urine, adding to each a small quantity of yeast, covering them with a saucer, and inverting them. If sugar be present in the urine, carbonic acid gas will collect at the upper part of that test-tube. A few bubbles of gas may come from the yeast itself, but the second test-tube containing water will show these also, so that any mistake is hardly possible. This test is not very sensitive. According to Roberts,* urines containing two grains and a half of sugar in the ounce, and under, yield no sign of sugar with this test.

Quantitative Estimation of Sugar.—A considerable number of methods have been devised for this purpose. I propose, however, only to describe two—viz., first, the modification of the process of Fehling which Dr. Pavy has recommended,† and, second, the method by means of the polarimeter.

Pavy's Method.—The principle upon which Fehling's method for the volumetric analysis of sugar depends is the reducing action which that substance has upon hydrated oxide of copper; but the reaction is so much obscured by the red precipitate of the sub-oxide which is thrown down that the results are not very accurate. Pavy therefore devised the following method, in which ammonia is made use of to prevent the precipitation of the sub-oxide. If ammonia be added to Fehling's solution, and the mixture be boiled, a sufficiency of grape sugar may be added to the mixture to reduce all the copper and render the solution colorless, without any precipitation taking place.

The preparation of the copper solution is carried out as follows: An ordinary Fehling's solution is made by dissolving 34.639 grammes of pure sulphate of copper in water and diluting to 500 c. c. The solution so obtained is mixed with another solution, prepared by dissolving 173 grammes of tartrate of potassium and sodium in water, mixing it with 100 c. c. of liquor sodæ (sp. gr. 1.34), and diluting the

* "Urinary and Renal Diseases," 4th ed., p. 183.

† *Proceedings of the Royal Society of London*, 1879.

mixture to 500 c. c. When these two solutions, each of 500 c. c., are united, we obtain one litre of ordinary Fehling's solution. Of this solution 120 c. c. are now taken, mixed with 300 c. c. of strong ammonia (sp. gr. .880), and diluted up to a litre with distilled water. This constitutes Pavy's standard solution, and of it 20 c. c. correspond to 0.01 gramme of grape sugar.

The analysis is carried out as follows: A flask of about 80 c. c. capacity is taken and fitted with a cork, through which two holes are bored, one of which receives the delivery tube of a Mohr's burette, and into the other is adapted a bent glass tube to allow of the escape of air and steam. The burette, filled with the urine,* is fixed in its stand, and the flask, into which 20 c. c. of the copper solution have been measured, allowed to hang free, so that nothing may obstruct the full view of its contents. Heat is now to be applied to the flask, and after the solution has boiled for a few minutes, so that all air has been expelled from the flask, the urine is allowed to flow into it until the copper solution has become completely colorless. This marks the end of the reaction. The quantity of urine used contains 0.01 gramme of grape sugar.

Method by Circular Polarization.—Grape sugar when in solution possesses this peculiar property, that if a beam of polarized light pass through it the beam becomes rotated to the right, and the degree of this rotation is in exact proportion to the amount of sugar contained in solution, and the length of the column of solution which the light traverses. Several instruments have been devised for the purpose of measuring the degree of this right-handed rotation, and so estimating the quantity of grape sugar present. Of these, the best known is the saccharimeter of Soleil-Ventzke. Its construction is complicated, and I do not propose to describe it in detail. It consists of two short brass tubes lying in line, and containing various polarizing prisms. Between these two end tubes fits in the tube containing the urine to be tested. By means of a milled head two quartz prisms are moved so as to compensate for the rotation effected by the sugar solution, and the amount of this movement is registered by means of an attached scale and vernier. When this scale stands at zero, and when no sugar solution is in

* It is best in the first instance to dilute the urine in the proportion of 10 to 100.

the tube, the appearance presented on looking through the instrument is a circular field divided into two lateral halves, each of which presents the same tint. If now the tube containing diabetic urine be slipped into its place, the light becomes rotated, and, on account of the special arrangements of the instrument, the field of vision assumes a different color on the two sides. By slowly moving the screw which commands the quartz prisms, these two colors become gradually altered in tint until they again exactly correspond to each other. The amount of movement required to effect this is now to be read off on the scale by means of the vernier, and by a simple calculation we can learn the percentage of sugar in the urine in question. With a tube one decimetre long each degree of the scale represents 1 gramme of grape-sugar in 100 c. c. of urine.

The urine must always be rendered perfectly clear by means of filtration before it is placed in the tube of the saccharimeter, and if it is highly colored it is well to remove the pigment by precipitation with acetate of lead and filtration. Albumen rotates polarized light to the left, as has been already mentioned, hence it is absolutely necessary to get rid of this substance, if it be present, before the saccharimeter is used.

Laurent's polarimeter is more accurate in its readings, and is to be preferred. The calculation is the same as that of the Soleil-Ventzke saccharimeter.

Saccharine urine is rarely met with except in cases of diabetes mellitus. Diabetic urine possesses, when the disease is fully developed, various well-marked characteristics. It is large in quantity, sometimes reaching so high a figure as 15 or 16 pints, and correspondingly pale, but nevertheless possesses a high specific gravity, ranging from 1040 to 1050, or even higher. The quantity of the nitrogenous substances excreted is usually, if not invariably, very much increased. The quantity of grape sugar excreted may, in severe cases, be as high as 25 or 30 ounces in twenty-four hours.

In certain cases of diabetes there may be felt in the urine, towards the termination of the case, a peculiar etherial odor, the result of the formation of *aceton;* and to the presence of this substance in the blood have been, by some, ascribed the symptoms of diabetic coma. Such urine usually assumes a reddish-brown color on the addition of chloride of iron, and this reaction is supposed to be due to the pres-

ence of ethyldiacetic acid, which, as Geuther and Gerhardt have pointed out, readily breaks up into aceton, alcohol, and carbonic acid. Much uncertainty, however, involves this interesting point.

Blood may be found in the urine as such (hæmaturia), or only blood pigment may be present (hæmoglobinuria); and these two conditions are readily distinguished by the fact that in the former case blood corpuscles are found on microscopic examination, while in the latter they are absent. The admixture of even a very small quantity of blood gives the urine a peculiar smoky appearance. When it is present in larger quantity the urine becomes bright-red or dark-brown. Small quantities of blood are best detected by means of the microscope, but when no corpuscles or crystals of hæmatin are present, recourse may be had to the spectroscope. If oxyhæmoglobin be present, two dark absorption bands will be seen lying between the lines D and E. On the addition of sulphide of ammonium to the specimen of the urine the spectrum of reduced hæmoglobin will appear—a broad dark band also lying between D and E, and less well defined than the bands of oxyhæmoglobin.

In cases of hæmaturia it is important to ascertain from what point in the urinary tract the blood comes, and this is not usually difficult. The hemorrhage may come:

(1.) *From the Urethra.*—The blood is mixed with the first portion of urine passed, often being expelled as a long clot, and it continues to flow in the intervals of micturition.

(2.) *From the Neck of the Bladder*, or prostatic part of the urethra. In this case the blood usually appears only at the very end of micturition, when the sphincter vesicæ begins to contract.

(3.) *From the Bladder.*—The blood is usually coagulated, and is passed in clots as large as the calibre of the urethra will allow to escape.

(4.) *From the Ureters.*—In this case the blood often appears in the form of long worm-like clots, which are casts of the ureters.

(5.) *From the Kidneys.*—When the blood comes from the kidneys it is uniformly diffused through the urine, is almost never in very large quantity, and when the urinary sediment is examined there are found tube casts, usually containing blood corpuscles.

Hæmoglobinuria appears in such diseases as purpura,

scurvy, pyæmia, severe typhus, small-pox, etc., and result from a breaking down of the red blood corpuscles in the blood stream, and the consequent liberation of the hæmoglobin they contain, which then escapes into the urine. It also occurs in a paroxysmal form, each paroxysm being accompanied with ague-like symptoms, and is then usually the result of a chill, although sometimes it may be traced to malarial infection.

Bile Pigment appears in the urine in cases of jaundice. Its presence is readily detected by the play of colors which ensues when the urine which contains the pigment comes in contact with nitric acid. If a little urine containing bile pigment is placed in a conical glass, and a few drops of nitric acid (which has been allowed to stand exposed to light for some time, and is therefore mixed with nitrous acid) are allowed to run down the edge of the glass and collect at the bottom of the vessel, a series of colored rings will form in the following order—yellow, violet, blue and green. A still better method is to filter the urine and then to allow a drop of nitric acid to fall on the surface of the filter. The play of colors ending in green will then be very distinctly seen.

Urine containing bile pigment is usually yellowish or greenish. It froths easily, and the bubbles of foam have a greenish-yellow color.

Bile Acids are found in the urine in considerable quantity in hepatogenic icterus. When to a solution of these acids a little cane sugar is added, and then a drop or two of sulphuric acid, a beautiful purple color develops itself. On this reaction depends their detection in urine. In a small quantity of urine a little sugar is dissolved, a strip of filter paper is dipped in, and then allowed to dry. If now a drop of sulphuric acid be allowed to fall upon the paper a purple ring will appear round it.

CHAPTER XXV.

URINARY SYSTEM—(*continued*).

URINARY SEDIMENTS.

IN order to examine the sediment of a urine it is best to allow the urine to stand covered for some hours in a conical glass, after which a drop or two of the sediment which has collected may be removed by means of a pipette and examined microscopically. It is of considerable importance to ascertain the reaction of the urine at the time it is depositing, and to note whether the specimen has been freshly passed or not.

Urinary deposits are divided into two classes—organic and inorganic. Of these the first is by far the most important.

Organic Deposits.

These include blood and pus corpuscles, epithelium, tube-casts, spermatozoa, and micro-organisms.

1. **Blood Corpuscles** are found in the urine in cases of hæmaturia. When the urine is acid the corpuscles preserve for some time their normal appearance; but when it is alkaline, or very dilute, the red corpuscles swell up, lose their biconcave shape, and become pale. On the other hand, when the urine is concentrated they shrink up and become crenated. It is very rare to find rouleaux of corpuscles. They are only seen in cases of profuse bleeding from the bladder. Occasionally crystals of hæmatoidin may be found.

2. **Pus Corpuscles** when present in any quantity form a yellowish-white deposit, which is usually easily recognizable by the naked eye. Microscopically, the corpuscles present as a rule their normal appearance; but if the urine be strongly alkaline, they tend to run together and form a homogeneous mass. If there be doubt as to whether a deposit consists of pus, it is only necessary to add a small piece of caustic potash, and to stir with a glass rod, when,

if the sediment be formed of pus, it will become tenacious, glassy, and semi-solid.

The presence of pus in the urine is always a sure sign that there exists an acute or chronic inflammation at some part of the urinary tract—renal abscess, pyelitis, cystitis, or urethritis. In women, it must be remembered, pus flowing from the genital tract may become mixed with the urine.

3. **Epithelium.**—The epithelial cells in the urine are best seen when stained with eosin or fuchsin. They may be derived from any portion of the urinary tract. The epithelium of the urinary tubules consists of round or polygonal cells, each having a large and sharply defined nucleus. Those of the pelvis of the kidney are conical, with one, or sometimes two, tail-like processes. The large irregular pavement epithelial cells which are often seen in the urine come from the bladder or vagina.

4. **Renal Tube-casts.**—Before examining the urine for tube-casts, a specimen should be allowed to stand in a conical glass for twenty-four hours, at the end of which time a few drops of the sediment may be raised by means of a pipette and examined microscopically (*vide* fig. 10). Staining with eosin or methyl-green will make the tube-casts more distinct.

Renal tube-casts* are almost invariably associated with albuminuria, and most frequently with Bright's disease, but they occasionally occur when no albumen can be detected in the urine. They are, as a rule, formed of fibrin, and are, as their name implies, casts of the renal tubules, in the majority of cases of the convoluted tubules of the cortex. The chief forms of tube-casts are the following:

(1.) *Epithelial Casts.*—In these the fibrinous cylinder has become covered over with epithelial cells which have been detached from the lining membrane of the tubule. These cells may be more or less cloudy and swollen. Such tube-casts are found in the inflammatory form of Bright's disease.

(2.) *Pus Casts.*—Casts containing pus corpuscles imbedded in them are sometimes met with. According to Dr. George Johnson, they are diagnostic of glomerulo-nephritis.

(3.) *Fatty Casts.*—Very frequently casts are found studded

---

*See an article by Dr. George Johnson, on the various forms of renal tube-casts.—*British Medical Journal*, March, 1882.

over with oil globules. These globules are the result of fatty degeneration of the renal epithelium, and such casts are met with in the second stage of inflammatory Bright's disease.

(4.) *Granular Casts.*—Dark opaque granular casts are also the result of epithelial degeneration in the renal tubules.

(5.) *Blood Casts* may either consist wholly of blood, the corpuscles being closely applied to one another, or fibrinous casts may be seen containing one or two blood corpuscles imbedded in them. Such casts point to capillary rupture, and are found in acute inflammatory Bright's disease.

(6.) *Hyaline Casts* are clear, homogeneous, and transparent, sometimes so delicate in structure as to be barely visible. They are for the most part formed in the convoluted tubules of the cortex, and have therefore a correspondingly convoluted form. The smaller specimens have been moulded within the lumen of a tubule which still retains its epithelium, while the larger varieties have been formed in tubules previously denuded of epithelium, and therefore of greater capacity. Hyaline casts point to chronic Bright's disease.

Occasionally hyaline casts may be found which exhibit the amyloid reaction, becoming reddish-brown on the addition of iodine, and dirty violet on the further addition of sulphuric acid; and giving also a beautiful violet with methyl-green, which tinges other casts green. Such waxy or amyloid casts are more strongly refractive than the ordinary hyaline variety, and being less flexible they exhibit deep fissures where they have been torn asunder in passing through the straight tubules.

The student should be careful not to mistake for tube casts these mucus coagula which are so often found, inclosing in their meshes whatever amorphous inorganic deposit the urine may happen to contain.

5. **Spermatozoa** are occasionally found in urine. They preserve their normal appearance for a long time. If the urine be very fresh, they may even be seen in active motion, but these movements are soon lost. Urine which contains spermatozoa becomes alkaline very rapidly.

6. **Micro-Organisms.**—Very many forms of lower organisms are found in urine. Many of these only develop after the urine has been voided, and are derived from the atmosphere. Occasionally, however, urine as it leaves the body

will be found to contain such organisms. These may be the result of the introduction of a catheter which has not been properly purified, but in other cases they are undoubtedly derived from the blood. The latter are most frequent in acute infectious disorders.* Roberts has described† a peculiar condition which he calls bacilluria, in which the urine when passed is opalescent from the presence of enormous numbers of bacilli. The reaction is acid, and when the urine has stood for some time the organisms sink to the bottom of the glass, leaving the supernatant fluid clear.

Very rarely the embryo forms of parasites which infest the blood are found in the urine.

Inorganic Sediments.

The reaction of the urine in which the sediment is found gives an important indication as to its constitution, certain substances separating out only in acid urine, while others are only found when the reaction is alkaline. The following table shows what the physician may be prepared to meet with in each case:—

Acid Urine.	*Alkaline Urine.*
Amorphous—	Amorphous—
Urates of potash and soda.	(*a.*) Neutral phosphate of lime.
	(*b.*) Carbonate of lime.
Crystalline—	Crystalline—
(*a.*) Uric acid.	(*a.*) Urate of ammonium.
(*b.*) Oxalate of lime.	(*b.*) Crystallized phosphate of lime.
(*c.*) Leucin.	(*c.*) Phosphate of magnesium.
(*d.*) Tyrosin.	(*d.*) Phosphate of ammonium and magnesium (triple-phosphate).
(*e.*) Cholesterin.	
(*f.*) Cystin.	

Sediments of Acid Urine.

1. *Urates.*—The amorphous deposit of urates, which is so frequently met with even in healthy urine, consists in the main of urate of soda, but may also contain urate of potash and of magnesia. To the naked eye the deposit of amorphous urates has a reddish, brick-dust color, due to pigmentation with uroerythrin. When the urine has been allowed

---

* Kannenberg, *Zeitschr., f. kl. med.*, i. p. 506.

† *Trans. of the International Medical Congress*, 1881.

to stand in a glass for some time, and deposit these urates, a peculiar bloom may be seen upon the sides of the glass when it is inclined, which is a characteristic and unmistakable sign of the presence of urates. Microscopically this deposit appears amorphous and finely granular. On warming the microscopic slide the sediment becomes dissolved, and it separates out again on cooling, and the same reaction can be very readily seen with a larger quantity in a test-tube.

In health a deposit of urates often occurs after profuse sweating and violent exercise, in cold weather. Pathologically this deposit is found in all febrile conditions, in grave organic disease, particularly of the liver, and in dyspepsia.

2. *Uric Acid.*—The more important points connected with the elimination of uric acid have been stated. There only now remain for consideration the appearances which a deposit of this substance presents in the urine.

The crystals of uric acid can usually be seen as bright reddish-brown grains adhering to the sides of the glass, or forming a layer at the bottom. They closely resemble the grains of cayenne pepper. Microscopically these crystals vary much in shape. They may take the form of four-sided tables, of six-sided rhombs, or they may be lozenge-shaped, ovoid, or barrel-shaped, or still more elongated and arranged in a stellate fashion. In whatever form uric acid appears the crystals are always more or less yellow; and as no other crystal which spontaneously separates out from urine is so tinted, there can be no difficulty in its recognition.

Fig. 10. Uric Acid Crystals and Tube-casts.

3. *Oxalate of Lime* appears in the urine as small octahedra, which may be more or less elongated, or in the form of dumb-bells or small ovoids. To the naked eye the deposit appears as a white, undulating, clearly-defined layer, resting upon a grayer deposit beneath.

Oxalic acid occurs normally in urine in minute quantities, and is much increased by the consumption of sugar,

and of such vegetables as contain it in their tissues. It constitutes one of the last stages in the decomposition of the effete tissues, and probably results from impeded metamorphosis (Beneke). Oxaluria is frequently produced by conditions which interfere with respiration and circulation.

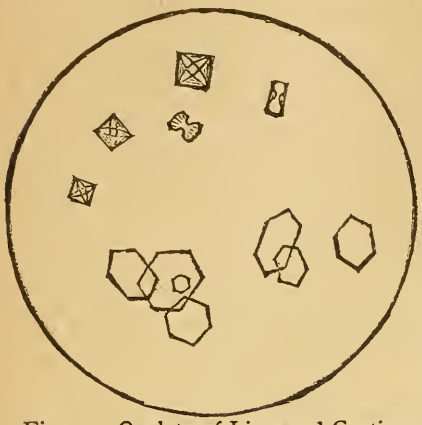

Fig. 11. Oxalate of Lime and Cystin.

As regards the symptoms of oxaluria, there is much difference of opinion, but it appears certain that in many cases at all events the appearance of oxalate of lime in quantity in the urine is accompanied with a well-marked train of symptoms. The patient is usually emaciated, very nervous, extremely hypochondriacal, and irritable. There is pain across the small of the back, a degree of irritability of the bladder, and general muscular weakness. It must be remembered, however, that, on the one hand, such a train of symptoms may be present in full development without any oxaluria, and, on the other, that crystals of oxalate of lime may be very abundant without producing any such symptoms.

4. *Leucin and Tyrosin.*—Leucin appears in the urine microscopically in the form of larger or smaller yellowish-brown balls, which sometimes show distinct striation. Tyrosin, on the other hand, appears under the microscope as sheaves of silky glittering needle-shaped crystals. These two substances result from the decomposition of albumen and other nitrogenous bodies, and when they appear in the urine they are a proof of incomplete oxidation of these substances, and they therefore precede urea. Leucin and Tyrosin are very rarely met with in urinary deposits. Their occurrence is almost confined to cases of acute yellow atrophy of the liver, and they are not by any means invariably met with in that disease.

5. *Cholesterin* and other fats are found in the urine in cases of chyluria. The deposit consists of minute oil globules, and when dissolved in a mixture of alcohol and ether, and the solution allowed to evaporate, clear plates of cholesterin often crystallize out.

6. *Cystin* appears in the urine in the form of six-sided

plates, which are insoluble in water and in acetic acid, but which readily dissolve in hydrochloric acid and ammonia. The pathology of cystinuria is very obscure.

Sediments of Alkaline Urine.

The inorganic sediments which are found in alkaline urine may consist of various salts of phosphoric acid, of carbonate of lime, or of urate of ammonium.

1. *Amorphous phosphate of lime* forms a whitish flocculent deposit, which is not dissolved by heat, but at once passes into solution on the addition of a drop or two of acetic or nitric acid. Under the microscope this deposit is seen to consist of fine granules, arranged usually in irregular groups. In microscopic appearance they closely resemble amorphous urates, but the reaction of the urine will at once indicate their nature.

2. *Crystallized Phosphate of Lime* (*Stellar Phosphate*).—The crystals of this salt are found in the urine in the form of rods, which either lie separately or are united to one another so as to form rosettes or sheaf-like bundles. This is a somewhat rare deposit, and is said to possess a graver significance than is attachable to the other varieties of phosphates.

Fig. 12.—Triple Phosphate and Stellar Phosphate.

3. *Phosphate of Ammonium and Magnesium* (*Triple Phosphate*).—This salt forms comparatively large clear crystals, which may frequently be recognized by the naked eye as bright sparkling points adhering to the sides of the glass. Examined microscopically, they are found to be of varying form, usually, however, having the shape of a triangular prism with bevelled ends, and presenting from above the appearance of a glass knife-rest. Most usually the deposition of these crystals is simply due to the ammoniacal decomposition of the urine.

4. *Phosphate of Magnesium* is occasionally, though rarely, encountered in the urine in form of crystals—flat tables elongated in shape, clear and glassy.

5. *Carbonate of Lime.*—In human urine carbonate of lime only occurs in an amorphous form. It dissolves in acetic acid with effervescence. In the urine of the horse it forms spheres marked with radiating lines.

6. *Urate of Ammonia* is found whenever the urine becomes strongly ammoniacal, in the form of opaque brownish spheres, which may be either smooth on the surface, or may be covered with minute spikes. Sometimes this salt crystallizes in the form of minute clear dumb-bells. It has no particular clinical significance.

CHAPTER XXVI.

Reproductive System.

The diseases of the male generative organs fall, almost without exception, within the domain of surgery. The only disorders with which the physician is called upon to deal are impotence and spermatorrhœa, and as their diagnosis does not, as a rule, present any feature of difficulty, I do not propose to consider them here.

Those diseases which are met with in connection with the female organs of reproduction usually receive the special study of the gynecologist, and are fully treated of in works devoted to that branch of medicine. Inasmuch, however, as very many of the disorders of the system generally which are encountered in women take origin in some disturbance of the generative functions, or are aggravated thereby, it is very important for the physician to possess some knowledge regarding their diagnosis. I shall, therefore, in very brief language, indicate the chief lines of inquiry in connection with

THE FEMALE REPRODUCTIVE ORGANS AND FUNCTIONS.*

Whenever it seems desirable to investigate the condition of these organs, we must first of all make some special inquiries into the past history of the patient, in addition to

* In this slight sketch of the subject I follow, with some abridgment, the method of case-taking adopted by Professor Simpson, in the Buchanan Ward in the Edinburgh Royal Infirmary.

those general points which have already been enumerated in Chapter I. The most important of these are as follows:

1. **Menstruation.**—In connection with menstruation we should ascertain—(1) the date at which it first appeared; (2) the regularity or irregularity of its return, and the interval of time between the commencement of one menstrual period and that of the next; (3) the duration of each period and quantity of the discharge; (4) the amount of pelvic or general disturbance which accompanies each menstruation; and (5) the date of termination, should the patient have ceased to menstruate.

If menstruation has become abnormal, then we may have to deal with one of three conditions—amenorrhœa, menorrhagia, or dysmenorrhœa.

(1.) *Amenorrhœa*, or absence of the menstrual flow, is a normal condition before puberty, after the menopause, and during pregnancy and lactation. Apart from these physiological states, the chief causes of amenorrhœa are the following:

A. Abnormal conditions of reproductive organs.
- (*a.*) Absence, atrophy or rudimentary condition of uterus and ovaries.
- (*b.*) Occlusion of the uterine or vaginal canal.
- (*c.*) Pelvic peritonitis.
- (*d.*) Cystic degeneration of both ovaries.

B. Abnormal hæmic conditions.
- (*a.*) Chlorosis.
- (*b.*) Anæmia.
- (*c.*) Debilitating diseases, as phthisis, etc.

(2.) *Menorrhagia.*—Under this term are included two conditions—first, increase of the menstrual flow; and, second, a flow of blood from the uterus during the intervals. The latter condition sometimes receives a special name—metrorrhagia.

Menorrhagia may result from various abnormal states of the system—plethora, anæmia, struma, constipation, Bright's disease, purpura, etc. It may also occur as the result of some local affection, such a polypus, fibrous or cancerous tumor, degeneration of the uterine mucous membrane, chronic ovaritis, etc., besides the various conditions—normal and abnormal—in connection with pregnancy, which give rise to hemorrhage.

When a constitutional cause can be clearly ascertained no local examination is necessary, but when this is not the case a careful physical examination should be made.

(3.) *Dysmenorrhœa*, or painful menstruation, may result from general or from local causes. The former include all those general conditions which predispose to or excite neuralgia, such as plethora, anæmia, malaria, gout, rheumatism, enervating habits, etc. Of local causes there are—(1) congestive and inflammatory conditions of uterus, ovaries, or pelvic cellular tissue; and (2) such conditions as lead to obstruction of the uterine or the vaginal canal. Unless a constitutional cause can be clearly ascertained, a careful local exploration ought to be made.

2. **Leucorrhœa,** or fluor albus—the white or yellow intermenstrual discharge so frequently met with—is of two varieties, according as it has its source in the uterus or vagina. In the former case it is alkaline and in the latter acid, and the vaginal form contains in much larger quantity those flattened and irregularly shaped epithelial cells which line that canal. Specular examination will at once show the source of the discharge in question.

3. **Pareunia.**

4. **Pregnancies.**—Former pregnancies have an important bearing on uterine disease. It is well, therefore, to note their number, the date of the first as well as that of the most recent, the state of health from conception to labor, the character of the labors, the patient's condition during the subsequent lactation, and finally, whether she has had any abortions.

Local functional derangements of the bladder, rectum, etc., and general disturbances of other systems, are considered elsewhere in these pages, and need not be repeated here.

PHYSICAL EXAMINATION.

The physical examination of the female generative organs comprises the following points:

1. **The Mammæ.**—As a sign of pregnancy the condition of the breasts is of considerable importance. The chief points are—enlargement and turgescence of the nipple,

deepening of the color of the areola, enlargement, hardness, and knotting of the glands themselves, together with the secretion of milk.

2. **The Abdomen.**—When treating of the physical examination of the abdomen, the diagnosis of uterine and ovarian tumors was postponed until we should come to consider the Reproductive System as a whole.

Ovarian cystic tumors are often very difficult of diagnosis. By the inexperienced such conditions as obesity, œdema of the walls of the abdomen, phantom tumor, or meteorism, may be mistaken for ovarian dropsy. But besides these and similar conditions in which the resemblance is somewhat remote, there are certain diseases which so closely resemble ovarian dropsy, that even experienced practitioners often find great difficulty in arriving at a diagnosis. The following table shows the main points of diagnostic difference between ovarian dropsy and ascites :

Ovarian Dropsy.	*Ascites.*
Tumor develops from one iliac fossa.	No history of lateral development.
Tumor is more ovoid when the patient lies on her back.	The bulging is at the flanks when the patient lies on her back, and the anterior abdominal surface is flattened.
On percussion the dulness is central, the intestines giving a clear note at the sides.	On percussion the dulness is at the flanks, to which the fluid gravitates; the centre of the abdomen, where the bowels float, being clear.
Change of position does not alter the line of dulness.	Change of position alters the line of dulness in the manner already described.
Œdema of the feet only occurs in the last stages.	Œdema of feet often occurs early in the case.

It occasionally happens that pregnancy is mistaken for an ovarian tumor. While all the ordinary signs and symptoms of pregnancy must be carefully considered, it is necessary to bear in mind that morning sickness and the mammary changes may occur in ovarian disease. Ballottement, the fœtal heart sounds, and the uterine souffle, are the most important diagnostic points. Even the passage of the uterine sound, and consequent abortion, would be better than the plunging of a trochar into the pregnant uterus under the belief that it is an ovarian cyst, an operation which

has been performed more than once by experienced surgeons.

The differential diagnosis between ovarian dropsy and fibrocystic tumor of the uterus is often a matter of very considerable difficulty. Dependence should be placed upon the slowness of the growth of the latter disease, and the enlargement of the uterine cavity, as well as upon the nature of the fluid obtained on tapping. Details will be found in special works on gynecology.

3. **The External Pudenda.**—The digital examination of the vulva will usually reveal any morbid condition which may exist in connection with the labia, the orifice of the urethra, the fossa navicularis, the vestibule, and the perineum. In this manner may be recognized, for example, inflammatory and suppurative processes in the labia, urethral caruncles, and, when the perineum has become destroyed, prolapse of the rectal or the vesical wall, or of the uterus itself.

4. **Examination per Vaginam.**—The patient should be laid on a couch or table on her left side, with the knees well drawn up and the shoulders low. The index finger of the left hand, previously well lubricated, is then to be gently introduced into the vagina and steadily passed upward to the *os uteri*. In this way information regarding the vulva and the vaginal walls will be obtained; and now the os and cervix may be carefully examined as to size, consistency, etc., and the finger or fingers (for it is sometimes advisable to introduce two) may be passed into the recto-uterine space, and finally swept along the roof of the cavity to the symphysis pubis. In this manner much may be learned as to the condition of the cervix and os, the position of the body of the uterus (retro- and ante-flexion and version), and the condition of the rectum, peritoneal sac, cellular tissue round the uterus, the bladder, etc.

Vaginal examinations should not be made in the case of unmarried women, unless the symptoms are severe.

5. **Bimanual Examination.**—Much more important than the ordinary vaginal examination is the bimanual examination, of which there are several varieties. While the fingers of one hand remain in the vagina, the other hand of the physician is used to bring the pelvic organs more fully within reach. The most usual position of the second hand

is on the anterior abdominal wall (abdomino-vaginal examination), the patient being made to lie on her back. Sometimes, however, the fingers may be passed into the rectum, while those of the other hand are in the vagina (recto-vaginal examination). At other times the passage of a sound into the bladder may be combined with vaginal and rectal palpation.

All the various possible varieties of this combined method place within our power a very complete examination of the pelvic organs. The uterus may be carefully palpated on all its surfaces, and its size, shape, and position clearly defined. Its consistence, sensitiveness, and mobility may also be ascertained. We may also thus learn the condition of the ovaries and fallopian tubes, and generally that of all the organs and important tissues contained within the pelvis.

6. **The Speculum** is much more useful to aid in treatment than in diagnosis. Its diagnostic value is confined to the inspection of the os and cervix and of the vaginal walls, which it allows of. Of the many varieties of this intrument which have been devised, the most useful are the cylindrical speculum of Fergusson, the bivalve of Cusco, and the duck-bill speculum of Sims. In using the two former instruments the ordinary lateral position usually suffices. The Sims speculum can only be advantageously used when the patient is placed in a special position, which is neither on left side nor on the face, but in a position between the two, the left arm being drawn out behind her, and the knees flexed—the right lying above the left. The Sims speculum requires to be held by an assistant. Whatever form of instrumeut be used it must be carefully warmed and oiled before introduction.

7. **The Uterine Sound** is an important aid to diagnosis. By its means we learn the size and mobility of the uterus, and the direction of the uterine cavity. The best variety of instrument is that devised by Sir James Simpson, more especially the modified form which Professor A. R. Simpson has recently recommended. The physician touching the os uteri with two fingers of the right hand, grasps the handle of the sound in the left, and passes the point of the instrument into the vagina, in close apposition to his finger, and thus guides it into the external os. If the uterus be normal

in position, the handle of the sound must now be moved round in a wide semicircle, so as to make the point look forward. The instrument will then glide into the uterine cavity. If, however, the uterus be retroverted this rotation will not be required. No force must be used in passing the sound.

Any displacement which exists will at once be recognized by the course which the sound takes, and the length of the uterine cavity can be gauged by noting how far the sound enters. To render this measurement more easy the instrument is marked at different distances from the point. At $2\frac{1}{2}$ inches (the normal length of the cavity) the sound presents a knob or ring, which is readily recognized by the finger in the vagina; at $3\frac{1}{2}$ inches there is a notch; at $4\frac{1}{2}$ inches a double knob or ring; at $5\frac{1}{2}$ a notch, and so on.

The uterine sound must not be introduced during a menstrual period, or when there is any suspicion of pregnancy, any inflammation of the uterus or in its neighborhood, or in cases of cancer of the cervix or body of the uterus.

8. **Instruments for Dilating the Cervix.**—Of these the most important are the various forms of tent. These instruments dilate the cervical canal, so as to allow the finger to pass into and to explore the uterine cavity. They are made of sponge, of sea-tangle, or of the wood of the tupelo or nyssa tree. The sponge tents expand more rapidly than either of the other kinds, but they are apt to induce fetor. The variety of tent which is to be selected in any given case depends upon the size of the cervical canal and the rapidity with which we wish dilatation to take place. The tent is most easily introduced through a speculum. The vagina should first of all be washed out with some antiseptic lotion, and then the tent, held in a pair of dressing forceps, gently introduced into the cervix. The tent should be lubricated with carbolized vaselin, and after it is in its place some carbolized cotton wool should be introduced into the vagina and packed up to the cervix. The tent should not be allowed to remain in the uterus after it has reached its full expansion, and in no case longer than twenty-four hours. After the introduction of the instrument the patient should be strictly confined to bed, and she should retain the recumbent posture for at least

twenty-four hours after its removal. Tents must not be employed when there is pelvic inflammation of any sort.

The cervical canal may also be dilated by passing into it a graduated series of metallic bougies, or, better, by the use of Molesworth's dilators, which consist of elongated bags of india-rubber, into which air or water can be forced by means of a powerful syringe.

Beside these methods of physical examination which have just been enumerated, it is necessary to mention the curette, by which a small piece of a tumor can be removed for microscopic examination; and the aspiratory needle, by which we can obtain a specimen of the contents of a doubtful cyst.

CHAPTER XXVII.

Nervous System.

SENSORY FUNCTIONS.

The diseases of the nervous system are so complicated, and at the same time their diagnosis is now becoming in the majority of cases so precise, as a strict logical deduction from the signs and symptoms observed at the bedside, that very special attention must be paid to the systematic arrangement of the inquiries which the physician has to make, and to the methods to be employed in testing the condition of the various functions of the nervous apparatus.

These functions will be considered in the following order :

1. Sensory Functions.
2. Motor Functions.
3. Vaso-motor Functions.
4. Trophic Functions.
5. Cerebral and Mental Functions.
6. Condition of Cranium and Spine.

SENSORY FUNCTIONS.

The phenomena met with in connection with the sensory apparatus are of two kinds ; firstly, *subjective, i. e.*, such sen-

sations as arise independently of any external irritation; and, secondly, *objective*, *i. e.*, sensibility to external stimulation of various kinds. Although this classification is open to considerable criticism, it will be found to be convenient for purposes of description, and for clinical examination.

Subjective Sensations.—Among the many sensations of which patients complain, the following are the most important :—

1. *Pain.**—Pain is simply an exaggeration of common sensibility. It is often the result of inflammation or other local disease of the tissues. When the pain is intermittent, darting, and occurs suddenly, following the course of some nerve and its branches, it is termed *neuralgia*. Of the many forms of neuralgia, the most commonly encountered are—

(*a.*) *Tic Douloureux*, neuralgia of the fifth nerve, which consists of paroxysms of pain corresponding in site to the nerve and its branches on the face.

(*b.*) *Intercostal Neuralgia* is, as its name implies, an affection of the intercostal nerves. It is to be carefully distinguished from the pain of pleurisy and of muscular rheumatism.

(*c.*) *Sciatica.*—The pain here corresponds to the course of the sciatic nerve and its branches. It is usually localized between the tuber ischii and the great trochanter, and shoots downward, sometimes as far as the heel.

(*d.*) *Visceral Neuralgiæ.*—These pains may be referred to the region of the heart and aorta (angina pectoris), of the stomach, intestines, liver, kidneys, uterus, or ovaries.

It was first pointed out by Valleix that, when the nerve which is affected with neuralgia lies superficially, there can be found during an attack certain points upon the skin, which are painful on pressure. Such painful points are usually situated where the nerve leaves an osseous canal or comes through a strong fascia.

Beside these neuralgiæ there are various painful sensations which are met with in nervous diseases, and which must be looked for in such cases. Of these we may notice—

(*a.*) *Girdle Pain*, which is the sensation of having a cord or girdle tied tightly round the body. It may be felt at

* Many of the varieties of pain mentioned are not subjective, arising as they do from local causes, but they are classified here because they occur without any stimulation of the sensory apparatus on the part of the physician.

various levels, on the thorax, abdomen, or on the lower extremities at the knee or ankle. It is commonly met with in connection with inflammatory and degeneration changes in the cord, and is believed to result from slight excitation of the posterior nerve-roots.

(*b.*) *Lightning Pains.*—Among the most common symptoms in locomotor ataxia, are paroxysms of darting, lancinating pains, which shoot through the body, and which have received the name of lightning pains.

(*c.*) *Headache* in all its many forms. Headache arises from a great variety of morbid conditions, such as vasomotor changes within the cranium, abnormal composition of the blood, organic disease of the cranium or scalp, or of the brain or its membranes. The differential diagnosis of these different forms is to be found in special works on the subject. It is sufficient now to point out that the mode of invasion, the intensity, and the site of the headache must be exactly ascertained, as well as the presence of any obvious exciting cause.

2. *Paræsthesiæ*, or perverted sensations, are commonly met with among disorders of the nervous system.

(*a.*) *Sensations of Heat and Cold* (independently of actual elevation or depression of temperature) are met with in intermittent fever, and also in various nervous diseases.

(*b.*) *Numbness.*—Any condition tending to depress the activity of the cutaneous sensibility may give rise to sensations of this kind, where the patient feels as if he were walking on a soft carpet, and has a tingling sensation up the limbs. It may also take the form of—

(*c.*) *Formication*, or the sensation of ants crawling over the skin. These two forms of paræsthesia are caused by affections of the nerve trunk (cold, mechanical injuries, etc.); or of the central organs (locomotor ataxia, hysteria, etc.), but probably not of the peripheral terminations.

(*d.*) *Pruritus*, or itching, is caused by disease or irritation of the terminal end organs in the papillæ of the skin. It arises as a result of—

(*a.*) Many skin diseases, particularly the parasitic varieties.

(*b.*) Prurigo.

(*c.*) Various chemical substances circulating in the blood—bile, sugar, hippuric acid, and perhaps xanthin and creatin (Eulenburg).

3. *Giddiness* (*Vertigo*), is a sensation of swimming in the

head, the body appearing to oscillate in different directions, and surrounding objects to rotate, and is accompanied with reeling and staggering. In Ménière's disease (auditory vertigo), and in disease of the cerebellum, vertigo is a frequent symptom ; but it also arises from any condition which disturbs the circulation in the cranium, such as dyspepsia, heart disease, mental work, etc., etc. In many cases vertigo arises from a contradiction between the impressions of external relations derived from two or more special senses.

4. *Abnormal Visceral Sensations.*—These comprise such sensory disturbances as pyrosis or water brash, boulimia or abnormal hunger, polydipsia or excessive thirst, and certain other similar symptoms.

Such are the more important of the abnormal sensations complained of by patients suffering from nervous disorders. We now turn to what is of much greater value in diagnosis, in that it admits of precise determination—viz., the actual condition of the cutaneous sensibility as tested by the physician himself.

Cutaneous sensibility is of two kinds—

(1.) *Common or General Sensation*, which includes the consciousness of contact with a substance which, when exaggerated, amounts to pain ; and

(2) *Tactile Sensation*, comprehending—

(*a.*) Sensations of pressure.
(*b.*) " " temperature.
(*c.*) " " locality.

These will each be considered in turn.

1. **Common Cutaneous Sensibility** may be tested by means of touching, tickling, pinching, or pricking the skin. All these methods of stimulation give indications, but by far the most accurate results, as Duchenne first showed, are obtained by using the Faradic current. The strength of the current may be varied from that just sufficient to cause a perceptible sensation up to one which produces sharp pain. By comparing the minimum strength of current required to cause a sensation at different parts of the body, important indications may be obtained.

2. **Tactile Sensibility** includes sensations of pressure, temperature, and locality.

(*a.*) *The Sense of Pressure* is best tested by the method devised by E. H. Weber, which consists in the application

of different weights over the portion of skin to be examined. In order to eliminate the muscular sense it is necessary to lay the hand or foot, as the case may be, upon a firm support, and further, it is advisable to interpose some non-conducting substance, so as to prevent the temperature or size of the weight from being recognized, as these impressions might give some indication of its weight. Other methods have been devised, but for all ordinary purposes that described is the simplest and best.

(*b.*) *The Sense of Temperature* is tested by applying to the skin two bodies of unequal temperature, the most convenient being two test-tubes, one filled with cold, the other with hot water. The most sensitive parts of the body are the skin of the face and of the back of the hands (Weber), where differences of temperature of 0.3 C. can readily be distinguished. A difference equal to a degree of the centigrade scale can be appreciated at any part of the body.

(*c.*) *The Sense of Locality* is to be tested by pinching some portion of the patient's body when his eyes are closed, and making him indicate the site of the touch. In health the error is very small indeed. It may further be tested by ascertaining to what distance the two points of a pair of compasses, or of Sieveking's æsthesiometer, pressed upon the skin, must be separated before they can be recognized by the patient as distinct. Weber gives the following as the minimum distances to which the points must be separated, to be felt as different points, in health:—

On the point of the tongue	1.18	m.m.
" palmar surface of last phalanx of finger	2.25	m.m.
" " " 2d " "	4.5	m.m.
" plantar " last " great toe,	11.25	m.m.
" back of the hand	31.5	m.m.
" forearm, and leg, and dorsum of foot	40.5	m.m.
" upper arm and thigh	77.5	m.m.

(1 m.m. = 0.039 inch.)

In disease all these various forms of cutaneous sensibility may be affected, either diminished (anæsthesia) or rendered more acute (hyperæsthesia), and they may be either collectively or individually affected.

When sensibility to pain is diminished, the condition is named *analgesia*, and when it is increased, *hyperalgesia*. In the same way, we speak of *thermo-anæsthesia* and *thermo-hyperæsthesia*, when the sensibility to heat is diminished or increased; and other refinements of nomenclature have been

devised by Eulenburg, such as *apselaphesia* and *hyperpselaphesia*, referring to tactile sensibility alone, the former indicating a decrease and the latter an increase. All these partial affections of the cutaneous sensibility are most frequently met with in cases of locomotor ataxia.

Finally, we have to note the rapidity of the conduction of these cutaneous sensory impressions to the brain. In health this takes place in a little over .1 second; and it is often easy—in the case of locomotor ataxia, for example—to observe an abnormal prolongation of this period. It is not uncommon to notice in such patients that the tactile and painful impressions do not travel at the same rate of speed, the prick of a needle being instantaneously felt as a touch, and, after an interval of a few seconds, as a painful sensation.

Muscular Sense *—that collection of impressions by means of which the mind is enabled to appreciate the degree of contraction of the muscles—demands careful investigation, since its presence or absence in a particular case is of great diagnostic significance. It is tested by causing the patient to lift various weights (suspended in a towel, so as to eliminate as far as may be the cutaneous sensibility to pressure), and we may either determine the smallest weight which can be appreciated as such, or the smallest difference between two weights which he can perceive. The muscular sense may also be tested by asking the patient, with closed eyes, to point to or to touch some particular part of the body, such as the tip of the nose or ear, or the great toe. The absence of this sense is frequently associated with loss of equilibrium of the body.

We now pass to the consideration of the special senses.

SIGHT.

For a full description of the various affections of sight, special works on the subject must be consulted. A very brief outline may, however, here be given of the alterations which arise as the result of nervous affections, it being distinctly understood that all defects of vision caused by abnormalities in the refracting media are excluded from consideration.

Optic Hyperæsthesia and *Hyperalgesia*, the increase of the

* The common sensibility of the muscles is not here alluded to, as being of minor importance and possessing little diagnostic significance.

retinal sensibility—amounting to a painful sensation, with intolerance of light—is common in meningitis and in cerebral hyperæmia, and illusions of sight often occur in insanity, dependent upon central changes. The most frequent pathological condition, however, is

Optic Anæsthesia, the diminution or total loss of vision. When (from nervous causes) the sight is merely impaired, we speak of *amblyopia;* when it is entirely lost, of *amaurosis.*

In *amblyopia* the vision may be affected as regards acuteness, extent, or color.

1. *Diminution in Visual Acuteness* causes the objects seen to appear blurred, misty, and indistinct. The most usual method of testing the acuteness of vision is by means of test types, which the patient is made to read at a distance. Defective vision may be merely functional, resulting from debility, gastric and uterine affections, abuse of tobacco, etc., when no abnormal ophthalmoscopic appearances present themselves; or it may be due to optic neuritis or retinitis, or to atrophy of the optic nerve.

2. *Alterations in the extent of the Visual Field* are most accurately determined by means of the perimeter, but, for all ordinary purposes, it is only necessary to stand opposite to and near the patient, who should be directed to shut one eye and to fix the other steadily on that of the operator. The latter should then move his hand in every direction, and ascertain at what points it enters the visual field. Any deviation from the normal condition can be thus readily detected. The visual field may be encroached upon either from the centre or from the margin. In the former case a dark spot (scotoma) forms in the centre and gradually enlarges. The scotoma may be steady, or scintillating, zigzag, and brightly colored, as in migraine. Such limitations of the field of vision are met with in functional amblyopia (especially from abuse of tobacco), in optic neuritis, and in optic atrophy.

The field of vision may further be abolished as regards one lateral half of the retinæ (hemiopia), the vertical line of demarcation being very sharply defined. This affection of vision is characteristic of disease of the optic tracts and commissure. The exact localization of the lesion can be deduced from the anatomical arrangement of the nerve fibres in the tracts and commissures, but for details special works must be consulted.*

* In particular, Charcot's "Leçons sur la Localisation des Maladies du Cerveau."

3. *Alterations in the Perception of Colors.*—To test the perception of color, a scale of colors as those of the spectrum should be employed, and the patient should be called upon to name them when presented to him. Color-blind persons cannot distinguish red. The visual field for the perception of colors may also be encroached upon. In health it is to be noted that the area of vision differs in the case of each color, being greatest for white—next yellow, then blue, red, and, last of all, green.

Movements of the Eyeball.*

The ocular muscles are supplied by three nerves—the oculo-motorius, or 3d nerve; the trochlearis, or 4th nerve; and the abducens, or 6th nerve. Spasm and paralysis of each of these affect the muscles which they supply, and produce corresponding changes in the position of the eyeball. Their action on the pupil will be subsequently considered.

I. Paralysis of the Ocular Nerves.

(1.) *Oculo-motorius, or 3d Nerve.*—According as the paralysis is complete or incomplete, the whole, or only one or more of the following muscles are affected:

(*a.*) *Levator Palpebræ Superioris.*—Paralysis causes drooping of the upper eye-lid—ptosis.

(*b.*) *Superior Rectus.*—The eyeball turns downwards and slightly outwards when this nerve is paralyzed, and there is in consequence diplopia or double vision, the result of the visual axis of the two eyes not being directed to the same object. As this divergence does not exist when the eyeballs are both turned downwards, the patient instinctively carries the head well thrown back.

(*c*). *Internal Rectus.*—Paralysis here gives rise to divergent strabismus (squint), with diplopia, the eyeball being rotated outwards on account of the unopposed action of the external rectus.

(*d.*) *Inferior Rectus.*—The affected eye is, in paralysis of this muscle, directed upwards and slightly outwards,

* Strictly speaking, these ought to be considered among the motor functions, but for convenience they are introduced here.

and there is diplopia except when the object lies above the level of the eyes.

(*e.*) *Inferior Oblique.*—In paralysis of this muscle the eyeball is turned slightly downwards and inwards, but this condition is rarely observed, as paralysis of the the inferior oblique as an isolated affection is exceedingly uncommon.

(*f.*) *Ciliary Muscle.*—The effect of paralysis of this muscle is that the patient is unable to change the convexity of the anterior surface of the lens, or, in other words, to focus his eye for near objects.

When the 3d nerve is paralyzed as a whole all these actions combine, and the result is that the lid droops, the eyeball is drawn downwards and outwards, and protrudes from its socket, the pupil (as we shall presently see) is dilated and immobile, and there is defective power of accommodation.

(2.) *Nervus Trochlearis, or 4th Nerve,* supplies the superior oblique muscle, and when that is paralyzed there is diplopia, the field of vision being moved downwards and outwards.

(3.) *Nervus Abducens, or 6th Nerve,* supplies the external rectus, paralysis of which causes convergent strabismus, with consequent diplopia, there being no power of rotating the eyeball outwards beyond the middle line. This condition frequently gives rise to giddiness, nausea, and vomiting.

Paralysis of the ocular nerves may be either central or peripheral. In the latter case the paralysis is usually more complete. Ocular paralysis is common in locomotor ataxia, syphilis, hemiplegia, etc., and after diphtheria.

II.—Spasm of the Ocular Nerves.*

(1.) *The 3d Nerve—*

(*a.*) *Levator Palpebræ Superioris.*—Tonic spasm of this muscle causes the eyelid to be drawn up, and the eye cannot therefore be closed. It results either from peripheral irritation (rheumatism, wound, etc.), or, reflexly, from irritation of the sensory fibres of the 5th nerve, or is due to central causes. Clonic spasm hardly ever occurs in this muscle.

* Muscular spasm is of two kinds—(1.) *Tonic*, when the contraction remains of nearly uniform intensity while the spasm lasts; and (2.) *Clonic*, when there is a condition of rapidly alternating contraction and relaxation.

(*b.*) *Internal Rectus.*—Of all the muscles of the eyeball this is the most frequently affected with tonic spasm. There is then convergent strabismus with diplopia, in which the two images are not at a fixed distance from each other (as in squint from paralysis of the 6th nerve), but undergo a constant oscillation, alternately approaching and receding from one another.

(2.) *The 6th Nerve.*—Tonic spasm of the external rectus is rarely met with without implication of other muscles. When present the eyeball is drawn outwards, giving rise to divergent strabismus.

Clonic Spasm of the muscles of the eyeball generally gives rise to a condition named *Nystagmus*, when the eyeball undergoes continuous oscillatory and rotatory movements which cannot be controlled by the will. It arises either from local abnormalities of the ocular structures, or from central nervous affections, such as locomotor ataxia, insular sclerosis, meningitis, hydrocephalus, etc. Coal-miners are frequently affected with nystagmus, owing to the constrained position and bad light in which they have to work. In them the nystagmus is probably strictly analogous to writer's cramp in clerks, and to trade spasms generally.

Changes in the Pupil.

The iris being under the control of two mechanisms, the one reflex (contracting)—the afferent fibres lying in the optic nerve, the efferent in the 3d nerve, and the centre in the corpora quadrigemina; and the other a dilating mechanism—the efferent fibres being in the sympathetic and the centre in the cilio-spinal region of the cord (lower cervical and upper dorsal); so spasm or paralysis of any of these structures will give rise to changes in the size of the pupils. There is probably, however, a third mechanism—viz., a local centre for the movements of the iris, lying either in the iris itself or in the neighboring choroid, and on this mechanism act various drugs when dropped on the conjunctiva, such as atropin (dilating) and physostigmin (contracting).

The most common causes of change in the pupils are as follows:

Contraction (myosis) occurs normally when the eye is accommodated for near objects, when the optic nerve or retina is stimulated, when the eyeball is turned inwards,

when the aqueous humor is defective, and under the influence of various drugs—morphia, physostigmin, etc. It may, further, be due to disease of the retina, or to irritation of the 3d nerve in its course, or of the higher nerve centres, to paralysis of the sympathetic, or to disease of the cilio-spinal region of the cord.

Dilatation.—Paralysis of the 3d nerve gives rise to dilatation of the pupil, and no movements of the iris are caused by exposure to light or by efforts at accommodation. Irritation of the sympathetic or of the cilio-spinal region may also cause dilatation of the pupil. Total paralysis of the iris may probably arise from disease of the lenticular ganglion (Hutchinson).

Hippus, or clonic spasm of the iris, shows itself in quickly alternating contraction and dilation of the pupil. It is sometimes seen in cases of paralysis of the 3d nerve, and probably in disease of the sympathetic.

Argyle-Robertson Symptom consists in the absence in the pupil of any response when the retina is stimulated by light, while at the same time it moves normally when accommodation is made. This condition is frequently met with in locomotor ataxia.

Ophthalmoscopic Examination should be practised by every student as frequently as possible, and should be a matter of routine in the examination of all nervous cases. Both the direct and the indirect methods should be used. When the fundus has been brought into view, we have to consider the condition of (*a*) the retinal vessels, (*b*) the optic nerve, (*c*) the retina, and (*d*) the choroid.

(*a.*) *Retinal Vessels.**—Note the size and arrangement of veins and arteries, and observe whether there be visible arterial pulsation (in cupping of the disc and in aortic incompetence), anæmia, hyperæmia, or hemorrhages.

(*b.*) *The Optic Nerve.*—Of abnormalities there may be—

(1.) *Simple Congestion of the Disc*—Vascularity; edges ill defined.

(2.) *Œdematous Congestion of the Disc*—Disc red; swollen; edges obscured.

(3.) *Neuritis*—Increased redness and swelling; the edges of the disc totally obscured.

---

* The arrangement of the morbid appearance of the fundus oculi here given corresponds pretty closely to that adopted by Dr. Gowers in his work on "Medical Ophthalmoscopy."

(4.) *Atrophy*—Disc white or gray; may be simple or secondary to congestion or to neuritis.

(*c.*) *The Retina.*—The chief abnormalities to be detected by the ophthalmoscope are hemorrhages, white spots, and patches.

(*d.*) *The Choroid.*—White spots occur, either new formations or the results of atrophy, with destruction of the pigment normally found there.

It may be convenient here to indicate shortly the ophthalmoscopic appearances in one or two diseases in which retinal changes are most frequent and important.

Cerebral Tumors.—In most cases optic neuritis is present. At first there is congestion, increased redness, swelling, and cloudiness in the disc; then neuritis sets in, with obscuration of the edges, followed by great swelling and strangulation of the papilla (choked disc).

Locomotor Ataxia.—Atrophy of the optic nerves is a frequent symptom in locomotor ataxia, and it is also sometimes seen in general paralysis, and occasionally in insular sclerosis. The disc is pale, small in size, and excavated, and the retinal vessels are usually diminished in calibre.

Bright's Disease.—Particularly in the cirrhotic form of this disease, special retinal changes (retinitis albuminurica) take place. These consist in (*a*) œdematous swelling of the retina; (*b*) white degenerative spots and patches; (*c*) small extravasations; (*d*) inflammation of the papilla; (*e*) consecutive atrophy of the nerve and retina.

Leucocythæmia.—In this disease the retina is pale, the veins dilated. Small extravasations of blood are frequent, and sometimes small white, lymphoid deposits are seen scattered over the retina.

HEARING.

Such affections of hearing as arise from pathological conditions in the external meatus, the Eustachian tube, and the middle ear, belong to the domain of surgery, and need not here be discussed. It is sufficient to point out that deafness arising from affection of the nervous apparatus may be distinguished from that caused by abnormalities of the conducting apparatus by means of the tuning-fork test. When a vibrating tuning-fork is placed on the vertex, the sound is heard most distinctly in the deaf ear if the deafness be due to defective conduction, because the waves of

sound, in their passage from the cranial bones outward through the meatus, are obstructed and thrown backward again and again on the internal ear. If, however, the defect of hearing arises from impairment of the auditory nerve, the sound of the fork will not be so distinctly heard on the deaf side.

Hyperæsthesia of the auditory nerves is common in hysteria, acute diseases, and insanity, and it may result from paralysis of the stapedius muscle, with consequent overtension of the membrana tympani in cases of facial paralysis. Auditory hyperæsthesia is frequently associated with various subjective phenomena, such as tinnitus and vertigo, the latter of which has been already considered. Anæsthesia of the sense of hearing may result from blows on the head, meningitis, cerebral and cerebellar disease, scarlatina, measles, typhus fever, and hysteria, and may be brought on by the administration of quinine. Deaf-mutism is most frequently congenital, the result of defective development of the internal or middle ear. When it is acquired, it is usually the consequence of some severe disease in childhood.

TASTE.

The sense of taste is located in the surface of the tongue, fauces, and back wall of the pharynx. The root of the tongue (circumvallate papillæ), fauces, and pharynx are supplied by the glossopharyngeal nerve. The taste nerve for the anterior two thirds of the tongue, on the other hand, is the lingual, and the majority, if not the whole of the sensory fibres of taste, pass from the lingual into the chorda tympani and then to the facial, which nerve, however, they leave below the geniculate ganglion, to join (by channels at present unknown) the fifth nerve, in the trunk of which they pass to the brain.

To test the sense of taste the patient should be made with closed eyes to protrude his tongue, on different points of which the substances in solution used in testing are to be deposited by means of a glass rod. For *bitter* tastes, solutions of quinine, picric acid, and infusion of quassia may be employed; for *sweet*, syrup is the most convenient; *acid* taste will be produced by the application of vinegar or dilute acids; and *saline* by means of solutions of common salt, bromide, or iodide of potassium. Sweet tastes are best felt

at the tip of the tongue, acid at the edges, and bitter at the root of the organ. Perhaps, however, the most accurate method of testing the sense of taste is by means of the constant current. Hyperæsthesia of the sense of taste is rarely met with, but it occurs occasionally in cases of hysteria. Paræsthesiæ or abnormal sensations of taste are sometimes met with in insanity.

Anæsthesia of taste may be peripheral, due to a coating of fur on the tongue, or abnormal dryness of the mouth, or to the action of heat or cold. It may also be due to defective conduction, from disease, of the nerves of taste in their course. In this way it may arise from lesion of the glossopharyngeal, when the defect of taste will be limited to the root of the tongue and fauces. When the anæsthesia involves the anterior two thirds of the tongue, it may be due to disease of the lingual nerve, of the chorda tympani, of the facial in the canal of fallopius, or of the fifth nerve within the cranial cavity.

SMELL.

The sense of smell is conveyed to the brain solely by the olfactory nerves. The branches of the fifth nerve distributed to the nasal mucous membrane have only to do with common and tactile sensation. To test the sense the patient must be made to smell various odoriferous substances, such as the essential oils, musk, camphor, valerian, etc.,* and to hold in the mouth such articles as cheese, wine, and liqueurs, which owe their agreeable flavor to the sense of smell (the latter test is particularly useful when the nostrils have become occluded). Hyperæsthesia of the sense of taste is occasionally seen in hysteria and general paralysis. Loss of smell may be due to any cause which prevents the access of the aromatic particles to the mucous membrane, such as polypus, catarrh, abnormal dryness of the membrane (in paralysis of the fifth), or paralysis of the muscles necessary to the act of "sniffing" from paralysis of the seventh nerve. But apart from these causes, there is a true anæsthesia of the olfactory nerve (anosmia), which occurs in hysteria, tumor of the brain, embolism of the middle cerebral artery, blows on the head, and, as Althaus has pointed out, in locomotor ataxia.

* Such substances as ammonia and acetic acid should be avoided, as they only irritate the branches of the fifth nerve, and not the olfactory.

CHAPTER XXVIII.

NERVOUS SYSTEM—(*continued*).

MOTOR FUNCTIONS.

FOR practical purposes, the various motor functions may be arranged in the following manner:

A. *Visceral Motor Functions.*
B. *Functions of Voluntary Muscles.*

I.—VOLUNTARY MOVEMENTS.
(1.) Paralysis.
(2.) Spasm.

II.—REFLEX ACTIONS.
(1.) Superficial reflexes.
(2.) Deep reflexes.

III.—CO-ORDINATION.

C. *Vaso-motor Functions.*

A.—Visceral Motor Functions.

The movements of the viscera are regulated by means of so complex a nervous mechanism, and enter as yet so little within the scope of diagnosis, that they need only be very briefly alluded to here. There are, however, certain reflex actions which are of diagnostic value as indicative of the condition of that part of the spinal cord where their centres are situated.

(1.) *Deglutition* has already been spoken of under the heading of the "Alimentary System." The reflex contractions of the œsophagus are in the main under the control of a centre in the medulla, and cease when that centre is diseased—as, for example, in advanced bulbar-paralysis.

(2.) *Micturition and Defœcation.*—The centres for these acts lie in the lumbar enlargement of the cord, and their performance is under the influence of the will. If from disease of the cord above the centres, volition is cut off, then, when the fæces have sufficiently accumulated, or the urine collected in the bladder to a certain amount, these excretions are expelled by means of reflex contraction. Involve-

ment of the sensory tracts in the disease prevents the patient from being conscious of these acts. When the lumbar centres are themselves affected, then fæces and urine are evacuated as soon as they enter the rectum or bladder.

(3.) *Sexual Functions.*—These functions are controlled by a reflex centre in the lumbar enlargement of the cord, so close to that for the cremasteric reflex that the condition of that reflex affords us a trustworthy indication of that of the sexual functions. If the control of the higher centres is cut off by disease in the upper part of the cord, the process becomes imperfect, and may sometimes be excessive (priapism). Disease of the sexual centre in the lumbar enlargement causes loss of sexual power.

(4.) *Respiration.*—The respiratory centre lies in the medulla close to and below the vaso-motor, the *nœud vital* of Fleurens. In advanced bulbar-parlysis, it is sometimes attacked with disease, with a necessarily fatal result.

B.—Motor Functions of Voluntary Muscles.

I. Voluntary Movements may be diminished, or they may be in excess. In the former case we speak of paralysis (akinesis) or paresis, according as the loss of muscular power is abolished or only diminished, and in the latter of spasm (hyperkinesis).

(1.) *Paralysis* (akinesis) of the voluntary muscles varies in its extent. It may be limited to one or two muscles, or it may affect all the muscles of one lateral half of the body (hemiplegia), or of both sides of the body symmetrically, usually both lower limbs (paraplegia).

To examine the state of the muscular system, it is necessary to cause the patient to go through all varieties of voluntary movement, simple and combined, standing, walking, stepping up upon a chair, etc., as well as such actions as speaking, writing, and the like, which require great accuracy and precision in the movement of the muscles brought into action. The dynamometer may be employed for ascertaining the force of the muscular contraction.

Electric Currents are of the utmost use in diagnosis, but the limits of this work prevent the various forms of apparatus being described. For such information the reader is referred to special works on the subject.* It will be suf-

* Such as Tibbit's "Medical Electricity," or Ziemssen's "Elektricität in der Medicin."

ficient here to indicate very briefly the inferences to be drawn from the information so obtained.

The *Faradic* or *Induced Current* excites muscular contraction when the stimulus is applied to the motor nerve in its course, or over the muscle itself. The contraction so induced varies from a scarcely perceptible change up to tetanus, according to the strength of current used.

The *Galvanic* or *Continuous Current* only gives rise to contraction when the current is opened or closed, not when it is passing. The reaction of each pole must be separately investigated, the other being placed upon the sternum.

The *Law of Normal Contraction* is as follows:

Weak Currents—

Positive pole (anode)—No contraction.

Negative pole (cathode)—Contraction when the current is closed, expressed by the formula C.C.C. (cathodal closing contraction); none when it is opened.

Currents of Medium Strength—

Positive pole—Slight contraction both on opening and on closing the current, expressed by the formulæ A.O.c. and A.C.c., anodal opening contraction and anodal closing contraction, the small c. indicating that the contraction is slight.

Negative pole—Strong contraction on closing the current, expressed by the formula C.C.C′., cathodal closing contraction, the accent on the last C′ indicating that the contraction is strong.

Strong Currents—

Positive pole—Contraction both on opening and on closing the current, expressed by the formulæ A.O.C., A.C.C.

Negative pole—Tetanus when the current is closed, slight contraction when it is opened, expressed by the formulæ C.C.Te., cathodal closing tetanus, and C.O.c., cathodal opening slight contraction.

Various forms of paralysis may be accurately classified, as Erb has shown, by means of the electrical reactions of the muscles and nerves, as follows:

a. No Change in the Electric Excitability with either form of current (cerebral paralysis, myelitis, rheumatic, and traumatic peripheral paralysis).

b. Quantitative Change in the Electric Excitability—

(*a.*) *Increase.*—This condition is uncommon, but is sometimes found in the first stage of cerebral hemi-

plegia, in certain forms of locomotor ataxia, and rarely and transitorily in peripheral paralysis at its commencement.

(β.) *Diminution* is rare in cerebral paralysis, but does sometimes occur in the later stages of labio-glosso-pharyngeal paralysis. It occurs in such spinal paralyses as are accompanied with muscular atrophy.

c. Quantitative and Qualitative Changes ("Reaction of Degeneration,"—*Erb.*)—The reactions of nerve and muscle are different, and must be separately stated as follows:

Nerves.

Two or three days after the paralysis has begun, the excitability of the nerves diminishes, and gradually becomes completely lost. Should recovery take place, the excitability reappears, but commonly it is later in being regained than voluntary motion.

Muscles.

To the Faradic current they behave much as the nerves do—the excitability being gradually lost, and as gradually regained on recovery taking place.

The galvanic excitability falls parallel with the Faradic for about a week, but in the course of the second week it begins to rise, until a point is reached when the muscles contract with stimuli which would have no apparent effect upon them in their normal condition. A qualitative change has also taken place, the positive closing contraction increasing until it equals or surpasses in intensity the negative closing contraction, while the negative opening contraction becomes equal to or exceeds the positive. By glancing at the normal law of contraction already given, it will be seen that in this form of paralysis the conditions are exactly reversed. After a time this galvanic excitability of the muscles diminishes, and in incurable cases disappear; but when recovery takes place, the normal state of matters becomes gradually restored. This "reaction of degeneration" occurs wherever peripheral nerves are affected in their course, and also in lead-poisoning, in poliomyelitis anterior acuta, and in progressive muscular atrophy.

(2.) *Spasm* (hyperkinesis) of the voluntary muscles, may be defined as abnormal muscular contraction, the result either of pathological irritation or of a physiological stimulus, to which the resulting contraction is disproportionate. It is of two varieties—tonic and clonic—the former indicat-

ing a condition of muscular contraction which remains of nearly equal intensity for a lengthened period (minutes, hours, or days), while under the latter term (clonic), is understood a condition of rapidly alternating muscular contraction and relaxation, whereby particular parts of the body are set in motion.

Of clonic spasms the simplest is *tremor*, which varies from the slightest fibrillary twitching of the lips or tongue to the most well-marked shaking of the limbs which paralysis agitans exhibits. Still more pronounced is the clonic spasm, which occurs in convulsions of all kinds (epileptic, uræmic, hysterical, etc.), in which the whole body may be violently tossed about by the muscular contractions.

Tonic spasm is most commonly seen as "cramp," continuous and painful contraction of muscles individually or in groups; also in catalepsy and in contracture (persistent shortening of muscle, owing frequently to changes in nutrition), and occasionally in the muscles used in certain coordinated movements.

In connection with spasm, it is to be noted that certain points are often to be found, pressure upon which either excites or arrests the spasm (motor exciting and motor arresting pressure points). This is particularly noticeable in connection with facial spasm.

II.—Reflex Movements of Voluntary Muscles.

The spinal cord has throughout its whole length numerous centres of reflex action. The stimulus enters the cord through the posterior nerve root, traverses the gray matter, and passes out again through the anterior roots, giving rise to muscular contractions. Many of the simpler reflex actions can readily be induced by the physician, and they are of great diagnostic value in that their persistence is an indication that no disease of importance exists in the reflex loop in question. Gowers* gives a very full description of the more important of these, and it is to be observed that, as we can originate reflex actions which have their centres at almost every point in the length of the cord, we can thus gain information regarding its condition throughout its whole extent. These reflexes are of two kinds—superficial and deep.

* "The Diagnosis of Diseases of the Spinal Cord," London, 1880.

Superficial Reflexes.

(1.) *Plantar Reflex.*—Tickling the skin of the sole gives rise to contraction of the muscles of the foot; centre in the lower part of the lumbar enlargement.

(2.) *Gluteal Reflex.*—Tickling the skin of the buttock determines in many persons a contraction of the gluteal muscles; centre probably at the level of the 4th or 5th lumbar nerves.

(3.) *Cremasteric Reflex.*—Tickling of the skin on the inner aspect of the thigh is followed by drawing up of the testicle; centre at the level of the 1st and 2d lumbar nerves.

(4.) *Abdominal Reflex.*—On stroking the skin of the abdomen from the costal margins toward the iliac crests, the abdominal muscles contract; centres lie between the 8th and the 12th dorsal nerves.

(5.) *Epigastric Reflex.*—Tickling the skin of the chest over the 4th, 5th, and 6th intercostal spaces, causes a dimpling of the epigastrium; centres from the level of the 4th to the 6th or 7th dorsal nerves. The same region of the cord contains centres for the reflex contraction of the erectores spinæ, which occurs when the skin is stroked from the angle of the scapula down to the iliac crest.

(6.) *Scapular Reflex.*—Tickling of the skin in the interscapular region gives rise to contraction of the scapular muscles; centre at the level of the lower two or three cervical, and the upper two or three dorsal nerves.

Deep Reflexes.

The afferent impressions which excite these have their origin in tendons or muscles—probably the latter. The chief of these are—

(1.) *The Knee Reflex.*—If the knee be flexed, and the leg be allowed to hang down loosely, a tap over the patellar tendon will give rise to a reflex contraction of the quadriceps femoris, and a consequent jerk forward of the leg. The centre for this reflex lies in the cord at the level of the 2d and 3d lumbar nerves. It is occasionally absent in health, about one per cent of individuals not exhibiting it.

(2.) *Ankle Clonus.*—In certain nervous conditions, a sudden pressure upon the sole of the foot which stretches the muscles of the calf, gives rise to a series of spasmodic contractions of these muscles, which recur with great regularity, usually about 5.7 times per second. The clonus can

also sometimes be excited by a tap on the muscles on the front of the leg. Similar reflexes may be obtained in the arm.

These reflexes are impaired by disease in any part of the reflex loop—sensory nerve, posterior nerve roots, gray matter of the cord, anterior nerve roots, or motor nerve. The patellar reflex is impaired and commonly lost in locomotor ataxia. It is excessive in disease of the antero lateral columns, and in hemiplegia on the diseased side. In this last-named condition the superficial reflexes are, on the contrary, impaired on the side affected.

III.—AFFECTIONS OF CO-ORDINATION.

Such complex muscular adjustments as are involved in standing, walking, etc., are probably co-ordinated in the cerebellum and higher nervous centres. They may be interfered with by any affection of the brain, spinal cord, or peripheral apparatus.

Labyrinthine Vertigo.—In Méniere's disease (disease of the semi-circular canals) inco-ordination of the muscular adjustments of the body may be observed leading to vertigo or to peculiar movements in particular directions, due to the fact that impressions of the position of the head, derived from the terminal nerve apparatus of the semi-circular canals, have failed or have become erroneous owing to disease of these canals.

Ataxia is that form of inco-ordination which accompanies disease of the spinal cord. It is most seen in the actions of standing and walking. The muscles do not move in harmony, the movements made being violent, jerky, and ill-directed. The heel is brought down to the ground suddenly and forcibly. The difficulty of movement is much increased when the eyes are shut, so that it is common to make such a patient stand with his heels together and with his eyes closed, in order to test his power of co-ordination. The ataxia then betrays itself in swaying and tottering movements. It is to be noted that the ataxia may be, and most usually is, associated with normal or even exaggerated muscular power.

Cerebellar Inco-ordination.—Affections of the cerebellum and neighboring ganglia may give rise to various symptoms of inco-ordination—reeling, vertigo, rigidity, and even sometimes to enforced movements, in which automatic

movements of the voluntary muscles are performed in spite of the will.

Loss of muscular co-ordination in connection with speech will be subsequently considered.

C.—Vasomotor Functions.

As an index of the state of these functions we have to take the condition of the skin as regards pallor or redness, temperature, and the amount of the various secretions. Such changes in the tissues as sloughing, etc., are rather to be referred to the trophic nerves.

I.—CUTANEOUS VASOMOTOR AFFECTIONS.

Diffused paleness or redness of the skin may be seen in persons in perfect health (blushing, etc.), but are often associated with nervous disorders such as epilepsy and hysteria, and may be induced by various drugs, as for example the flushing which follows the inhalation of nitrite of amyl.

In fever the vasomotor nerves of the skin appear to be in a condition of abnormal irritability. The hot, cold, and sweating stages of ague seem to depend upon general tonic contraction of the vessels of the skin, followed by general relaxation, originating in all probability from the centre in the medulla. More local changes may be brought on by mechanical or chemical irritation, but sometimes occur independently of such, as for example the local and circumscribed vasomotor epileptic aura, which Nothnagel has described. Further, in various neuroses (epilepsy, Graves' disease, etc.), there are to be found scattered over the skin of chest and abdomen red blotches of congestion, to which Trousseau gave the name of *tâches cerebrales*, and which may sometimes be excited by drawing a pencil point over the skin. Various affections of the central nervous system are followed by vasomotor changes in the skin. In paraphlegia the temperature of the paralyzed limbs frequently undergoes an increase, which is followed by a diminution when the peripheral nerves are affected. In cases of cerebral hemiplegia the temperature in the paralyzed parts is almost invariably slightly elevated, and remains so for some time. Ultimately, however, it falls again, and in old standing cases not only is the temperature on the affected side lower

than that on the healthy, but the pulse is smaller and more compressible, and the hand and foot pale and cold. Unilateral sweating and other vasomotor disturbances are not uncommon in Graves' disease, epilepsia, and hysteria.

Visceral Vasomotor Affections.—Lesion of the brain and medulla, and even passing psychical disturbances, often determine vasomotor changes in internal organs (congestion of viscera in hemiplegia, disorders of menstruation from emotions, etc.). The secretions are often affected from such causes. The urine, in particular, is liable to well-marked quantitative and qualitative changes, injury of the 4th ventricle and other areas in medulla, cerebellum, and cord, giving rise to polyuria, albuminuria, and glycosuria. It seems also probable that certain forms of enlargement of the liver and spleen are dependent upon vasomotor changes determined by affections of the central nervous system.

CHAPTER XXIX.

NERVOUS SYSTEM.—(*continued*).

TROPHIC FUNCTIONS OF THE NERVOUS SYSTEM.

AMIDST a great deal that is uncertain in respect to these functions, there are several well-ascertained facts which may be briefly alluded to here. The nutrition of all the tissues appears to be under the control of the nervous system. Even the lower portion (cord, nerves) of that system itself are, as the descending degenerations show, controlled from the higher centres. One or two of the more important of these trophic changes may be mentioned—

I. *Muscular Tropho-neuroses.*—Muscles may atrophy as the result of local causes, or as a consequence of long inaction (such as follows paralysis from cerebral hemorrhage or embolism). Apart from these causes, when active neurotic atrophy of the muscles occurs, it is due either to disease of the nerve cells in the anterior horns of gray matter in the spinal cord or corresponding regions in the medulla, or to affections of those nerve fibres which connect these cells with the affected muscles. It is in such cases that electrical examination gives the "reaction of degeneration" already de-

scribed, and hence its great importance as a means of diagnosis.

II. *Affections of Bones and Joints.*—In all nervous affections, whether peripheral or central, changes in the nutrition of the bones and joints are liable to occur. Amongst the best known are those which follow locomotor ataxia, which may be acute articular swelling, closely resembling rheumatism, or chronic degenerative changes, leading to great deformities and strong predisposition to fractures. The important point to note in regard to these affections is that they are unaccompanied with pain.

III. *Affections of the skin.*—So far as is yet known, the nerve fibres connected with cutaneous nutrition leave the cord, along with the posterior sensory nerves, and it appears probable that they spring from the cells of the posterior horns of the cord. Various eruptions, such as erythema, urticaria, eczema, herpes, may arise as the result of disease of these nervous structures, and Paget has described an affection of the skin of the fingers, "glossy skin," which is due to the same cause. Still more important, as belonging to this category, are the acute and chronic bedsores, which are so common and so troublesome in spinal cases. And, finally, there have to be noted the occurrence of pigmentation, and of affections of hair, nails, and cutaneous secretory apparatus, and the more profound lesions of lepra anæsthetica, all of which must be included among the cutaneous tropho-neuroses.

IV. *Affections of the Secretory Glands.*—Salivation and lacrymation, as well as the flow of the bile and other secretions, are under the influence of the nervous system, but do not give diagnostic indications further than has been already noted in other parts of this work.

V. *Affections of the Viscera.*—Of these too little is known to aid in diagnosis.

CHAPTER XXX.

Nervous System—(*continued*).

CEREBRAL AND MENTAL FUNCTIONS.

In many diseases of the nervous system the intellectual powers are affected. The powers of attention and memory

of the patient are put to sufficient proof while the physician is informing himself regarding his history. Decadence of the power of judgment may betray itself in connection with the business transactions of the patient, which, if obviously irrational, will usually be communicated by his friends. The most obvious interference with the intellectual powers is, however, loss of consciousness, or coma.

Coma is met with in simple fainting, in injuries of the head, in apoplexy (in which case it is accompanied with paralyses), in epilepsy (usually accompanied with convulsions), in hysterical attacks, in catalepsy, in uræmia, in severe attacks of fever of various kinds, and in narcotic poisoning. It is sometimes very difficult to establish a diagnosis between alcoholic poisoning, for example, and apoplexy, The state of the pupils, the smell of the breath, the condition of the heart, and the presence or absence of paralysis, will, however, usually make clear the nature of the case. Coma vigil, in which the patient lies unconscious with the eyes wide open, is met with in cerebral diseases.

There are certain other disorders of intelligence which frequently occur in mental disease, and which must be noticed here.

Illusions are objective disorders of perception—a sound is heard, or an object seen; but both perceptions are misinterpreted.

Hallucinations, on the other hand, are subjective disorders of perception. The patient may imagine, for example, that rats are running over the bed-clothes, or that he hears people calling to him, when no foundation exists for either belief. These are hallucinations. But if he observe some dark object on the bed and take it to be a rat, that would be an instance of an illusion.

Delusions have no relation to perception. They are purely mental, and are only met with in the insane. It is not uncommon, for example, to meet with such delusions as that the patient believes himself to be the Deity.

Delirium, or wandering of the mind, indicated by incoherent speech, may consist in low muttering or in wild and furious shouting. The former variety is most frequently met with in cases of nervous exhaustion, the result it may be of fever, or of any grave organic disease. The more noisy form of delirium occurs in meningitis, in acute mania, and as the result of some poisonous ingredient circulating in the blood, such as alcohol, fever poison, belladonna, car-

bonic acid, and other substances. Delirium may also be caused by reflex irritation in connection with such organs as the stomach or uterus. A variety is not uncommon in pneumonia, and is even occasionally met with in phthisis pulmonalis.

It is also important to note further the condition of the patient in regard to mental *emotions*, whether these are under full control or not. This is very obviously not the case in hysterical persons, and in many other nervous affections the patient may be observed to be emotional and excitable.

Speech.—The physiology and pathology of speech are of a most complex nature, and can only be touched upon here. We must first of all shut out all imperfections of speech due to laryngeal affections, which are grouped under the head of aphonia, and which are apart from the present subject.

In order that an individual may employ language as a means of communicating with his fellows, he must first of all be able to understand their gestures or speech—the receptive and regulative department of speech; and, secondly, he must be able to express his own mental state by means of gestures or speech—the emissive and executive department.

The *receptive* function is performed by means of the various end organs of special sense, and by the nervous apparatus which connects these with the central organs. Persons who are born blind, or become both blind and deaf, are examples of impairment of the receptive function of speech.

The *regulative* function of language, or that whereby the individual reduces to order and to comprehension the impressions which reach his brain, is impaired in the disease known as amnesic aphasia. This affection of speech takes many different forms. Sometimes the patient cannot remember the names of things, while every other part of his sentences comes easily to him. Sometimes he cannot name an object held up in front of him, while he can talk and write fluently. At other times the initial letter of the word may be all that he can recollect. Finally, much more pronounced cases are often met with, in which the patient cannot understand spoken or written language at all, and in which his own words are meaningless. The lesion in cases of amnesic aphasia corresponds to the distribution of the

posterior and terminal branches of the middle cerebral artery.

The *emissive* function of language—viz., that by which the speech impulses are co-ordinated before being sent to the executive department, is impaired or lost in the disease known as ataxic aphasia. The patient understands perfectly what is said to him or what he reads, and he has no difficulty in articulation, but he is unable to communicate his thoughts by speech or by writing (agraphia). His speech consists of what have been called "recurring utterances," which may or may not chance to be appropriate to the occasion, and which do not possess any intellectual value. The lesion is usually in the third left frontal convolution or in the island of Reil.

The *executive* department of language, that, namely, which regulates articulation, is impaired in diseases of the medulla oblongata (bulbar paralysis), and in general paralysis of the insane. In the former affection the speech is "scanning," that is, the words are uttered syllable by syllable. In the latter the syllables are misplaced, and there is stuttering and stammering.

For details regarding the pathology of the function of language special works must be referred to. Enough has been given here to show how any particular case is to be classified.

Sleep.—The disorders of sleep are of considerable practical importance. They are mainly three—

1. *Somnolence.*—Apart from the natural aptitude for sleep possessed by persons of a lethargic temperament, the causes of somnolence are mainly as follows: Exposure to external cold, especially when combined with insufficient nutriment; overloading the stomach with food; dyspepsia; blood poisoning (uræmia, fevers, poisoning with narcotics, alcohol, carbonic acid, etc.); disease of the brain.

2. *Insomnia*, or want of sleep, may be directly due to pain. It may further arise from excessive mental work, worry, anxiety, from dyspepsia, from the use of tea or coffee, from cerebral disease, from insanity, and from heart disease.

3. *Somnambulism.*—In this case also a definite cause, similar to the above, may usually be found.

CHAPTER XXXI.

Nervous System—(*continued*).

CONDITION OF CRANIUM AND SPINE.

Cranium.—The condition of the cranium sometimes affords important indications in nervous cases. Thus in epilepsy we may find a distinct localized depression, the result of an old depressed fracture, and which may indicate the cause of the disease. In chronic hydrocephalus, again, the peculiar shape of the skull is globular, the frontal bones prominent, and the eyeballs protruding; the sutures are open, and the fontanelles large and pulsating. Occasionally, also, tumors of the cranial bones may be detected which may have given rise to symptoms of compression.

Spine.—Examination of the spine includes inspection, palpation, percussion, and the "hot sponge test."

Inspection.—The patient should, if practicable, be stripped, and be made to stand upright, with the feet close together and firmly planted. If an ink mark be made on the skin over the tip of each spinous process, the line of the spine will be rendered distinct, and any lateral curvature will readily be detected. At the same time, any displacement of the column caused by angular curvature will become apparent.

Palpation of the spinal column should be practised both posteriorly and anteriorly through the abdominal walls. Tumors of the vertebræ can thus be detected.

Percussion of the spine posteriorly causes pain when there is disease of the vertebræ, or of the spinal membranes, in myelitis, in spinal irritation, etc.

Hot Sponge Test.—This test consists in passing down the spine a sponge which has been wrung out of warm water, and which is not so hot as to be unpleasant to the healthy skin. In certain cases, particularly in myelitis, pain is experienced by the patient as the sponge passes over the seat of the disease.

CHAPTER XXXII.

Locomotory System.

BONES—JOINTS—MUSCLES.

Bones.—In cases of suspected syphilis, the surface of the bones, particularly the cranium and tibia, ought to be carefully examined to detect the presence of nodes. The softness of the osseous tissues generally in cases of mollities ossium, and the enlargement of the articular ends of the bones, and their altered shape in cases of rickets, are also to be looked for.

Joints.—An examination of the joints must be made whenever pain is complained of in them, or when the presence of pyæmia, rheumatism, or gout is suspected. Allusion has already been made to the joint affections which are met with in locomotor ataxia. Hysterical joint affections are more surgical than medical in their nature. The acute swelling and inflammation of the joints in rheumatic fever and in gout is usually very obvious. In rheumatoid arthritis, also, the joints become enlarged, deformed, and stiff, movement being accompanied with distinct crackling.

Muscles.—The condition of the muscular system has already been to a large extent considered under the head of the nervous system. The following points, however, demand further notice:

Rigidity of muscles is due to irritation of the motor nerve tissue. This sometimes occurs in spastic paralysis, in meningitis, and sometimes in hysterical paraplegia.

Contracture, or persistent shortening of muscles, may be due to paralysis of their opponents (secondary contracture), to cirrhosis of the muscular tissue (myopathic contracture), or to abnormal innervation (neuropathic contracture).

Flaccidity occurs when the muscle has become atrophied.

Fibrillary twitchings of the muscular fasciculi occur in many spinal affections, but are most marked in progressive muscular atrophy.

Muscular paralysis, spasm, and atrophy have been already considered.

APPENDIX A.

In how far, from an examination of the fæces, the physician may infer what is the exact condition of the intestinal tract, is a question of some difficulty. The following points, which may be of use in localizing catarrhal affections of the intestines, are, in great measure, derived from an article by Professor Nothnagel (*Zeitschrift für Klinische Medicin*, vol. iv. 1882, p. 223), which appeared after the second chapter of this work was in type, and which could not, therefore, be interpolated in the text.

The evacuation of pure mucus from the bowel without any admixture of fæces points to catarrh of the rectum. When firm fæces are passed, completely enveloped in mucus, we may conclude that the morbid process affects the lower part of the colon and the rectum. The admixture of mucus with the fæces in abnormal quantity is not always apparent to the naked eye. It often happens that when the fæculent matter is examined microscopically there are found scattered intimately through it small masses of mucus, which are whitish-gray hyaline and transparent. This peculiar admixture of mucus indicates that the catarrhal affection is limited to the upper portion of the large intestine (and, possibly, the small intestine), while the rectum and descending colon are free from disease. When the stools contain small masses of mucus tinged yellow with bile pigment, we may conclude that the small intestine has become affected. In normal fæces, the reaction which is characteristic of bile pigment cannot be obtained; but when, from whatever cause, the peristaltic action of the small intestine is increased, this characteristic play of colors will be seen on the addition of nitric acid.

APPENDIX B.

Method of Preparing the Solution of Nitrate of Mercury used for the Determination of Urea.

This standard solution is best prepared from pure mercury. Of this substance 71.48 grammes are to be carefully weighed out, placed in a large beaker-glass, and treated with five times that weight of pure nitric acid of specific gravity 1.425. The solution so obtained is to be warmed in a water bath until all the nitrous acid vapor has been driven off, and the solution has become colorless. A few drops more of the acid are to be added until nitrous fumes cease to develop themselves, the solution evaporated to the consistence of syrup, and then carefully diluted with water up to one litre. The accuracy of the solution of nitrate of mercury so obtained must now be tested by comparing it with a standard solution of urea, prepared in the following manner: Two grammes of pure urea, dried *in vacuo* over sulphuric acid, are dissolved in water and diluted until the fluid has a volume of exactly 100 c.c. Of this solution 10 c.c. contain 200 milligrammes of urea. By using the volumetric method detailed in the text, the standard solution of urea being substituted for the urine, the exact strength of the solution of nitrate of mercury prepared may be readily ascertained; and it can, if necessary, be altered in concentration by adding more mercury or more water, as the case may be, so as to bring it to the exact strength mentioned in the text—viz., 1 c.c., corresponding to 0.01 grammes of urea.

INDEX.

A.

Abdomen, auscultation of, 60.
examination of, 42, 59, 60.
form of, 43.
inspection of, 43–45.
movements of, 45.
palpation of, 46–52.
percussion of, 53–60.
prominence of, 43.
pulsation in, 45.
retraction of, 44.
tumors in 45.
walls of, 41.
Abdominal aorta, pulsation of, 52.
organs, auscultation of, 60.
reflex, 252.
Abdomino – vaginal examination, 263.
Abducens, nervus, paralysis of, 241.
Abscess of throat, 32.
Accentuation of heart sounds, 86.
Aceton, 216.
Acetonæmia, 192–246.
Achorion Schönleinii, 182.
Acidity of stomach, 31–37.
Acid fermentation of urine, 194.
Acne, 178, 179, 180.
Acuteness of vision, 239.
Addison's disease, bronzing in, 19.
Adenia, 63.
Adenitis, simple, 62.
syphilitic, 62.
strumous, 62.
Adherent pericardium, 72, 73.
Aegophony, 175.
Aesthesiometer, 237.
Agraphia, 259.
Ague, feeling of cold and heat in, 235.
perspiration in, 20.
temperature of, 24.
vaso-motor affections in, 254.
Air cells, epithelium from, in sputa, 125.
Air columns in lung influencing percussion, 149–155.
Akinesis, 248.
Alar thorax, 136.
Albumen in sputa, 121.
in urine, detection of, 207.
estimation of, 208.
varieties of, 207
Albuminuria, 210.
transitory, 210.
Alcoholic poisoning, 257.
Alimentary system, 25–60.
Alkaline fermentation of urine, 195.
Alopecia areata, 208—footnote.
Amaurosis, 239.
Amblyopia, 239.
Amenorrhœa, 227.
Amnesic aphasia, 258.
Amphoric resonance, 160.
respiration, 168.
Amyl-nitrite, inhalation of, 254–109.
blushing caused by, 18.
Anacrotic pulse, 106.
Anæmia, blood in, 65–68.
blood pressure in cases of, 117.
breathing in, 138.
color of urine in, 190.
leading to amenorrhœa, 227.
menorrhagia in, 227.
œdema in, 20.
of larynx, 132.
pallor of, 18.
pallor of gums in, 28.
lips in, 26.
palpitation in, 70.
pulse curve of, 109.
specific gravity of urine in, 194.

Anæsthesia, 237.
of taste, 245.
optic, 239–245.
Analgesia, 237.
Aneurism, aortic, 70–75–101.
aortic, causing paralysis of recurrent laryngeal nerve, 133.
cough of, 121.
dulness of, 28.
of abdominal aorta, 52.
Animal parasites of skin, 185, 186.
Ankle clonus, 252.
Anode, 249.
Anorexia, 36.
Antero-lateral columns, disease of, knee reflex increased in, 253.
Anthrax, 66.
Aorta, abdominal, aneurism of, 52.
thoracic, dulness of, 82.
Aortic aneurism, causing paralysis of recurrent laryngeal nerve, 134.
area, 84.
disease, auscultation of arteries in, 98.
diastolic murmurs, 93.
regurgitation, sphygmographic tracing of, 93.
cardiographic tracing of, 114.
causing capillary pulse, 75.
retinal pulsation in, 243.
systolic murmurs, 92.
valves, 83.
Apex-beat, 71.
tracings of, 112.
Aphasia, amnesic, 258.
ataxic, 258.
Aphonia, causes of, 129.
Apices, percussion of, 160.
Apoplexy, 257.
Appetite, depraved, 36.
derangements of, 36.
excessive, 36.
loss of, 36.
Argyle-Robertson symptom, 243.
Argyria, 20.
Arteries, atheromatous, 95.
auscultation of, 98.
examination of the, 95.
inspection of, 95.
murmurs in, 99.
palpation of, 95.
Arteries, sounds of, 99.
Arterioles, spasm of, causing cyanosis, 18.
Arytenoid muscle, paralysis of, 134.
Ascaris lumbricoides, 42.
Ascites, 43, 47, 53.
distinguished from ovarian disease, 229.
respiration in, 138, 140.
Asthma, round shoulders in cases of, 23.
sputa of, 127.
Ataxia, 253.
Ataxic aphasia, 258.
Atheroma, 95, 106, 108.
Atrophy, acute, of liver, leucin and tyrosin in urine of, 224.
of optic disc, 243.
of muscles, 255.
Atropine, dryness of mouth after administration, 29.
Attention, 257.
Attitude, 23.
Auditory nerve, anæsthesia of, 245.
hyperæthesia of, 245.
Auscultation, areas for, 84.
auscultation respiratory system, 163.
of abdominal organs, 60.
of arteries, 98.
of heart, 83, 94.
of œsophagus, 35.
of veins, 101.

B.

Bacilluria, 222.
Bacillus anthracis in blood, 65.
Barrel-shaped chest, 137.
Basal tone of percussion, 147.
Bile acids in urine, 218.
Bile pigment in urine, 218.
Bilious constitution, 22.
Bilirubin, relations with uro-bilin, 190.
Bi-manual examination, 263.
Bladder, catarrh of, mucus in urine in, 212.
gall, 49, 55.
urinary, 59.
Blebs, 180.
Blindness, 239.
color, 240.

Blood, estimation of hæmoglobin in, 68.
examination of the, 64–69.
filaria, in, 66.
in fæces, 41.
in sputum, 123.
in urine, 216.
in vomited matter, 39.
micro-organisms in, 65.
microscopic examination of the, 64.
pigment granules in, 65.
pressure, estimation of, by sphygmomanometer, 117.
Blood corpuscles,—
alterations of, 65.
enumeration of, 66.
in urine, 218.
Blue line on gums, 28.
Blushing, 254.
Boiling test for albumen in urine, 207.
Boils, 180.
Bones, condition of, 261.
nodes on, 261.
trophic affections of, 255.
Bothriocephalus latus, 42.
Boulimia, 36.
Bowels, 50, 60.
cancer of causing constipation, 40.
Brain, disease of, *see* cerebral disease.
Breathing, *see* respiration.
Breasts, 229.
Bright's disease, albuminuria in, 210.
blood-pressure in, 117.
dry skin of cirrhotic form, 21.
frequency of micturition in, 188.
menorrhagia in, 227.
œdema in, 20.
ophthalmoscopic appearances in, 244.
pallor of, 18.
of lips in, 26.
quantity of urine in, 189.
skin affection in, 181.
specific gravity of urine in, 193.
tube casts in, 220.
urea diminished in, 198.
Bronchi, obstruction of, vocal fremitus in, 142.
Bronchial casts in sputa, 126.
catarrh, dry râles in, 173.
harsh breathing in, 165.
sputum in, 122.
respiratory murmur, 166.
Bronchiectasis, sputum of, 123, 124, 126.
Bronchitis, cough of, 97.
sputum of, 123, 124.
Bronchocele, 63.
Bronchophony, 174.
Broncho-vesicular respiration, 169.
Bronzing of skin, 19.
Bruit de diable, 101.
de pot fêlé, 159.
see, also, Murmurs.
Bubbling râles, 172.
Bubo, 62.
Bulbar paralysis, electricity in, 249.
laryngeal paralysis in, 133.
palate in, 33.
salivation in, 31.
speech in, 259.
swallowing affected in, 35.
tongue in, 29.
Bulging of thorax, 138, 144.
Bullæ, 180.

C.

Cadaveric position of vocal cords, 133.
Calculus in bladder, pain caused by, 188.
renal, pain caused by, 188.
biliary, *see* Gall-stone.
Callipers, 144.
Cancer of bowels, causing constipation, 40.
kidneys, 52.
lymphatic glands, 63.
œsophagus, 35.
omentum, 51.
pallor in cases of, 18.
pancreas, 50.
stomach, 36.
tongue, 29.
Capillaries, defective filling of, 18.
dilatation of, 18.
state of, 100.
Capillary pulse, 75.
Carbolic urine, 191.
Carbonate of lime in urine, 226.
Carcinoma, *see* Cancer.

Cardiac auscultation, 83, 94.
murmurs, 87.
pain, 69.
percussion, 80–82.
pulsation, 71–76.
region, 71.
sounds, 86.
Cardiograph, 111.
curve, normal, 112.
Caries of teeth, 28.
Carotid pulsation, 73.
Casts of bronchi, 126.
rental tubules, 220.
Catalepsy, 251, 257.
Cathode, 249.
Cavernous breathing, 168.
Cavities, air, modifying percussion, 158.
in lung, amphoric resonance in, 160.
bronchial breathing over, 168.
cracked-pot sound in, 159.
metallic tinkling in, 172.
vocal resonance over, 175.
Cephalic murmur, 100.
Cerebellar inco-ordination, 254.
Cerebellum, disease of, vertigo a symptom of, 236.
Cerebral affections, paralysis of tongue in, 29.
hyperæmia, intolerance of light in, 239.
and mental functions, 256.
paralysis, electric conditions of, 249.
tumors, ophthalmoscopic appearances in, 244.
Cervical veins, 101.
Cervix uteri, dilatation of, 265.
Chancre, soft, 62.
hard, 62.
Chest, see thorax.
Chest-measurer, 145.
Cheyne-Stokes breathing, 139.
Chlorides in urine, estimation of, 202.
quantity excreted, 203.
Chlorosis, 101.
cardiac pain in, 70.
depraved appetite in, 36.
gastric pain in, 36.
leading to amenorrhœa, 227.
pallor of, 18.
Chlorosis, pallor of lips in, 26.
venous murmur in, 102.
Cholera collapse, expression in cases of, 21.
pinched lips of, 26.
Cholesterin in urine, 224.
Chorea, movements of tongue in, 29.
Choroid, appearance of, 244.
Chylous urine, 62–66.
Cicatrices, 180.
Cilio-spinal region of cord, 243.
Circulatory system, 69–118.
Cirrhosis of liver, vomiting of blood in, 39.
Clonic spasm of voluntary muscles, 250.
Clonus, ankle, 252.
Coalminer's nystagmus, 242.
Cold and heat, sensitiveness to, 235.
Color blindness, 240.
of urine, 190.
perception of, 240.
Coma, 257.
vigil, 257.
Comedo, 179.
Common sensation, 236.
Condensation of lung tissue causing bronchial breathing, 167.
Conduction of sensory impressions, rapidity of, 237.
Consciousness, loss of, 257.
Consolidation of lung, dry râles in, 173.
resonant râles in, 173.
tympanitic note in, 158.
vocal resonance in, 175.
Constipation, 40.
Constitutions, 22.
Continued fever, 24.
Continuous current, action on muscles, 249.
Contraction, law of normal, 249.
of pupil, 242.
Contracture muscular, 251–261.
Co-ordination, affections of, 253.
Corpuscles, blood, alterations of, in form, 65.
variations in number, 65.
enumeration of, 66.
Cough, 120.
its frequency, 121.
its character, 121.

Counter-irritants, effects of, on skin, 211.
Cracked-pot sound, 159.
Cramp, 251.
Cranium, condition of, 260.
Creatinin, 200.
Cremasteric reflex, 248–252.
Crepitant râle, 170.
Crico-thyroid muscle, paralysis of, 133.
Crisis, 24.
Croup, 132.
breathing in cases of, 138.
Crusts, 180.
Crystals in sputa, 127.
asthma, 127.
Curette, 266.
Currents, electric, use of in diagnosis, 248.
medium effects of, 249.
strong effects of, 249.
weak effects of, 249.
Curvature, spinal, 260.
Cutaneous vaso-motor affections, 254.
Cutis anserina, 179.
Cyanosis, 18.
of lips, 26.
Cyrtometer, 144.
Cystin in urine, 205–224.
Cystitis, pain of, 188.
pus in urine in, 219.

D.

Deafness, 244.
Deep reflex, 252.
Defæcation, 40–247.
Degeneration, reaction of, 250.
Deglutition, 34–247.
interference with, 34.
Delirium 257.
tremens, 257.
tremulousness of lips in, 27,
Delusions, 257.
Demodex folliculorum, 187.
Dentition, 27.
Desquamation, 180.
Development, 17.
Diabetes, characters of urine in, 216.
color of urine in, 190.
dry skin of, 20.
dryness of tongue in, 30.
excessive appetite in, 36.
Diabetes, frequency of micturition in, 188.
odor of urine in, 192.
quantity of urine in, 189.
saliva in, 31.
specific gravity of urine in, 193.
thirst in, 36.
urea increased in, 198.
Diarrhœa, 40.
Diastolic aortic murmur, 93.
heart-sound, 83-84.
mitral murmurs, 91.
Diathesis, 22.
Dicrotic notch, 105.
pulse, 106–109.
Dilatation of aorta, dulness in, 81.
altering second sound of heart, 86.
of cervix uteri, 265.
of pupil, 242.
of right auricle increasing cardiac dulness, 82.
Diphtheria, 32.
ocular paralysis after, 241.
paralysis of larnyx in, 133.
paralysis of palate in, 32.
paralysis of pharynx in, 34.
throat in, 33.
Diplopia, 240.
Disc, *see* optic disc.
Displacements of uterus, 265.
Diuresis, 189.
Dropsy, *see* ascites and œdema.
Ducts, thoracic, 61.
Dynamometer, 248.
Dysmenorrhœa, 228.
Dyspepsia, faintness in, 36.
pallor of, 18.
palpitation in, 70.
saliva in, 31.
tongue in, 29.
vertigo a symptom of, 236.
Dyspnœa, 140.
attitude in cases of, 23.
breathing through mouth in, 27.
heat, 138.

E.

Eating, sensations after, 36.
Eclampsia, spasm of tongue in, 29.
Eczema, fissures in, 181.
marginatum, 185, footnote.

Eczema, papulosum, 179.
scales of, 180.
Electric current, use of in diagnosis, 249.
Embolism, cerebral, tongue in, 29.
Emotions, mental, 258.
Emphysema, pulmonary, breathing in, 139–141.
diminishing cardiac dulness, 81.
displacing liver, 56.
apex beat, 76.
enlargement of thorax in, 137.
inspiratory pressure in, 147.
œdema in, 19.
percussion note in, 155.
percussion of apices of lung in, 156, 161.
round shoulders in, 23.
subcutaneous, 20.
Encephalic murmurs, 100.
Endocardial murmurs, 87.
thrills, 78.
Enlargement of thorax, 137–144.
Enumeration of blood corpuscles, 66.
Epigastric pulsation, 74.
reflex, 252.
Epiglottis, paralysis of, 133
Epilepsy, 257.
albuminuria, after an attack of, 211.
convulsions of, 251.
depressed fracture, causing, 260.
spasm of tongue in, 29.
vaso-motor affections in, 254.
Epileptic convulsions, 251.
Epithelium in sputum, 125.
in urine, 220.
Equilibrium of body, 238.
Eructation, 37.
Eruptions, skin, distribution and configuration of, 177.
elements of skin involved in, 178.
etiology of, 181.
type of, 178.
Erythema fugax,
nodosum, 178.
Eustachian tubes, orifices of, 129.
Excoriations, 180.
Exocardial murmurs, 94.
Exophthalmic goitre, 64–95.
albuminuria in, 112.
palpitation in, 153.
prominence of eyeballs in, 21.
Expectoration, *see* sputa.
Expiration, prolongation of, in vesicular respiration, 165.
Expiratory pressure, 147.
vesicular murmur, 164.
Expired air, measurement of, 145.
Expressfon of face, 21.
Eyeball, movements of, 240.
Eyelid, drooping of, 240.
Eye, muscles of, 239.

F.

Face, expression of, 21.
Facial paralysis affecting palate, 33.
interfering with mastication, 33.
Fæcal tumors, 51.
Fæces, 40.
parasites in, 42.
blood in, 41.
Fainting, 70.
Faintness, 36.
Falsetto voice in recurrent paralysis, 133.
Family history, 16.
Faradic current, action on muscles, 249.
Fasting, sensations during, 36.
Fat, 17.
in urine, 256.
Fauces, examination of, 32.
ulceration of, 32.
Favus, 181.
crusts, 180.
Fehling's test for sugar in urine, 213.
Female reproductive organs, 226.
Fermentation of urine, 195.
test for sugar in urine, 214.
Ferro-cyanide test for albumen in urine, 208.
Fever, albuminuria in, 210.
attitude of patient in cases of severe, 23.
breathing in, 138.
color of urine in, 190.
delirium of, 257.

Fever, deposits of urates in urine, 223.
diminution of urine in, 189.
excretion of chlorides in, 203.
excretion of phosphates in, 206.
hectic, 21.
intermittent, 25.
loss of appetite in, 36.
pulse-tracing in, 111.
recurrent, 25–66.
relapsing, 25.
remittent, 25.
sordes on lips in, 26.
specific gravity of urine in, 193
temperature in, 24.
tongue in, 29.
urea increased in, 198.
uric acid, excretion in, 200.
uro-bilin in urine of, 190.
vaso-motor affections in, 37.
Fibrillary twitching, 251.
Fibrin in urine, 210.
Field of vision, 239.
Filaria sanguinis hominis, 66.
First sound of the heart, 84.
Fissures, 80.
at angle of lips in syphilis, 23–26.
Flaccidity of muscles, 261.
Flatulence, 38.
Flea-bites, 187.
Fluctuation in ascites, 48.
in thorax, 143.
Fluor albus, 228.
Foreign bodies in larnyx, 132.
Form of thorax, 136.
Formication, 235.
Fourth nerve, paralysis of, 241.
Fremitus, vocal, 142.
Friction distinguished from râles, 174.
Friction, pericardial, 79–94.
pleural, 143–173.
Fundamental tone of percussion, 148.
Fur on tongue, 30.

G.

Gall bladder, palpation of, 50.
percussion of, 55.
Gall-stones in fæces, 42.
causing vomiting, 38.
Galvanic current, action on muscles, 248.
Gastric pain, 37.
ulcer, pain of, 37.
vomiting of blood in, 39.
General paralysis of insane, speech in, 259.
tongue in, 29.
Giddiness, 235.
Girdle pain, 234.
Gland, thyroid, 63.
thymus, 64.
Glands, trophic affections of, 256.
lymphatic, 62.
inflammation of, 62.
syphilis of, 62.
cancer of, 63.
ductless, 63.
Globulin in urine, 208.
Glottis, œdema of, 132.
narrowing of, cracked-pot sound in cases of, 159.
Gluteal reflex, 252.
Glycosuria, 212.
Goitre, 63–64.
exophthalmic albuminuria in, 210.
prominence of eyeballs in, 21.
paralysis of recurrent laryngeal nerve in, 134.
Goose-skin, 179.
Gout, gastric pains in, 37.
uric acid excretion in, 200.
Gouty constitution, 22.
Grape sugar in urine, 213.
Graphic methods, 103.
Grave's disease, vaso-motor affections in, 255.
Grayness of skin, 20.
Gums, 28.

H

Habits, 16.
Hæmacytometer, 67.
Hæmatemesis, 39.
distinguished from hæmoptysis, 123.
Hæmatin, relations with uro-bilin, 191.
Hæmaturia, 217.
Hæmic murmurs, 94.
Hæmoglobin in blood, estimation of, 68.
Hæmoglobinuria, 217.

Hæmoptysis, distinguished from hæmatemesis, 123.
Hair, falling out of, after syphilis, 23.
Hallucinations, 257.
Harsh vesicular respiration, 165.
Headache, causes of, 235.
Hearing, affections of, 244.
Heart-burn, 37.
Heart, auscultation of, 83–94.
fatty, pallor in, 18.
inspection, 71–75.
murmurs, 87.
normal curve of, 112.
palpation, 76–79.
percussion, 81–82.
position of, 79.
sounds, 83–84.
enfeeblement of, 86.
impurity of, 86.
intensification of. 86.
modifications of, 85.
reduplication of, 87.
valves of, 83.
Heart disease, albuminuria in, 210.
ascites in, 54.
breathing in, 138.
cyanosis in, 18.
diminution of urine in, 189.
specific gravity of urine in, 193.
subcutaneous œdema in, 20.
vertigo a symptom of, 235.
Heat and cold, sensations of, 235.
Hectic fever, malar flush in, 21.
temperature in, 24.
Height, 17.
Hemi-albumose in urine, 209.
Hemicrania, redness of skin in, 19.
Hemidrosis, 20.
Hemiopia, 240.
Hemiplegia, 248.
knee reflex increased in, 252.
superficial reflexes diminished in, 253.
tongue in, 29.
vaso-motor affections in, 255.
Hepatic dulness, 54.
Herpes zoster, distribution of, 178.
pain of, 119.
Heteromorphism of thorax, 137.
Hippocratic succussion, 176.
History, 16.
Hodgkin's disease, 63.
Hot sponge test, 129.
Husky voice in laryngeal affections, 129.
Hydatids of liver, 49.
Hydrocephalus, chronic, shape of skull in, 260.
squint in, 21.
Hydro-chinon, 191.
Hydrogen, sulphuretted, in urine, 204.
Hydrophobia, salivation in, 31.
Hydro bilirubin, 191.
Hyperæsthesia, 238.
optic, 238–245.
Hyperalgesia, 237.
optic, 238.
Hyperdicrotic pulse, 110.
Hyperidrosis, 20.
Hyperpyrexia, 24.
Hypertrophy, of left ventricle, pulse tracing of, 110.
blood pressure in, 117.
causing increase of dulness, 82.
of heart, altering position of apex beat, 76.
increasing strength of apex beat, causing increase of dulness, 80.
Hysteria, 257.
breathing in, 139.
color of urine in, 190.
convulsions of, 251.
cough in cases of, 120.
dyspnœa, 140.
joint affections in, 261.
spasm of tongue in, 29.
spasm of œsophagus in, 35.
vaso-motor affections in, 254.

I.

Ichthyosis, 179.
Icterus, *see* jaundice.
Illusions, 257.
Incontinence of urine, 248.
Indican in urine, 201.
Induced current, action on muscles, 249.
Inferior laryngeal nerve, paralysis of, 133.
Inflammation of lungs, *see* pneumonia.

Inflammation of lymphatic glands, 62.
of lymphatic vessels, 61.
redness of skin in, 19.
Injuries, previous, 23.
Insomnia, 259.
Inspection of abdomen, 43–45.
arteries, 95.
circulatory organs, 71.
liver, 45.
nares, 128.
respiratory system, 135.
stomach, 44.
veins, 101.
Inspiratory pressure, 147.
vesicular murmur, 164.
Integumentary system, 176–187.
subjective phenomena. 177.
objective phenomena, 177.
eruptions, 177.
Intellectual powers, 256.
Intensity of percussion sound, 149-153.
Intercostal neuralgia, 234.
pain of, 119.
distinguished from pleurisy, 119.
Intermittent fever, 25.
Internal thyro-arytenoid muscle, paralysis of, 134.
Intestinal obstruction, indican excretion in, 202.
Intolerance of light, 239.
Iris, *see* pupil.
Itching, 235.

J.

Jaundice, 19.
yellow sweat in, 21.
uro-bilin in urine of, 190.
Jerk, knee, 252.
Jerky respiration, 165.
Joints, affections of, in hysteria, 261.
in rheumatism, 261.
trophic affections of, 255.
Judgment, 257.

K.

Kidneys, disease of, causing ascites, 54.
floating, 58.
palpation of, 51.
percussion of, 58.
Kidneys, tumors of, 45–51.
Knee reflex, 252.

L.

Labia, examination of, 230.
Labio-glossal paralysis, *see* bulbar paralysis.
Labyrinthine vertigo, 253.
Language, 259.
Laryngitis, cough of, 121.
Laryngoscopic examination, 130.
Larynx, anæmia of, 131.
catarrh of, 132.
changes in color, 132.
croup of, 132.
examination of, 129.
with laryngoscope, 131.
foreign bodies in, 132.
movements of, 132.
nerves of, 133.
palpation of, 129.
paralysis of individual muscles of, 134.
stenosis of, breathing in, 138.
tumefaction of, 132.
ulceration of, 132.
Lead-poisoning, albuminuria in, 211.
blue line on gums in, 28.
paralysis of, 250.
Lenticular ganglion, disease of, 243.
Leprosy, 179.
Leucin in urine, 224.
Leucorrhœa, 228.
Leucocythæmia, 63.
blood in, 65.
breathing in, 138.
ophthalmoscopic appearances in, 244.
spleen enlarged in, 49.
uric acid excretion in, 200.
Lice, 180.
Lichen pilaris, 179.
Light, intolerance of, 239.
Lightening pain, 235.
Lips. color of, 26.
form of the, 26.
movements of the, 27.
Liver, acute yellow atrophy of, leucin and tyrosin in urine, 220.
cirrhosis of, 39–48.
causing ascites, 54.

Liver, diminution of, 57.
diseases of, causing jaundice, 19.
diseases of, uric acid execretion in, 200.
displacement of, 56.
enlargement of, 56.
examination of, 45.
hydatids of, 49.
outline of, 54.
palpation of, 48.
percussion of, 54.
effect of respiration on, 55.
size of, 49.
shape of, 55.
surface of, 48.
tenderness of, 48.
tumors of, 45–49.
tumors of, causing bulging of thorax, 137.
waxy, 48.
Locality, sensation of, 237.
Locomotor ataxia, affections of cutaneous sensibility in, 237.
affection of sense of smell in, 246.
electric conditions of, 249.
knee reflex lost in, 253.
ocular paralysis in, 241.
ophthalmoscopic appearances in, 244.
tongue in, 29.
trophic affections of joints in, 255.
Locomotory system, 261.
Logwood, urine colored by, 191.
Louse, 187.
Lung, air in, influencing percussion, 153–155.
auscultation of, 163.
apices, percussion of, 160.
cavities in, amphoric resonance in, 160.
amphoric respiration over, 168.
cracked-pot sound in, 159.
percussion of, 158.
vocal resonance over, 175.
cirrhosis of, shrinking of thorax in cases of, 138.
collapse of, crepitant râle in, 170.
Lungs, consolidation of, causing bronchial respiration, 167.
dry râles in, 173.
resonant râles in, 173.
tympanitic note in, 159.
vocal resonance in, 175.
regional percussion of, 161–162.
relaxation of, cracked-pot sound in, 159.
rupture of, causing sub-cutaneous emphysema, 20.
tissue in sputa, 126.
relaxation of, causing tympanitic note, 157.
tension of, influencing percussion, 155.
tumor of, causing enlargement of thorax, 137.
vibration of air in, on percussion, 148.
Lupus, 178–179.
Lymphatic constitution, 22.
vessels, inflammation of, 61.
narrowing of, 61.
dilatation of, 61.
rupture of, 62.
glands, 62.
inflammation of, 62.
syphilis of, 62.
cancer of, 63.
Lymphangiectasis, 61.
Lymphorrhagia, 61.
Lysis, 25.

M.

Macules, 178.
Malar flush, 21.
Malarious fever, blood in, 65.
Mammæ, 228.
Mania, delirium of, 257.
Mastication, 33.
Measles, desquamation of, 180.
temperature of, 25.
Measurement of tracings, note on, 117.
Mediastinal tumors, 101.
Medicines, skin eruptions caused by, 181.
Melanæmia, 65.
Melanin, 191.
Memory, 257.
Ménière's disease, 236–253.

Meningitis, delirium of, 257.
excretion of phosphates in, 206.
intolerance of light in, 239.
Menorrhagia, 227.
Menstruation, 227.
Menstruation, respiratory system, 143.
Mental emotions, 257.
blushing from, 18.
pallor from, 18.
Mental functions, 257.
Mercury causing looseness of teeth, 28.
salivation, 31.
Mesenteric glands, tumors of, 51.
Metallic tinkling, 172.
Micro-organisms in blood, 65.
in sputa, 127.
in urine, 221.
Microscopic examination of blood, 64.
fæces, 42.
sputa, 124.
vomited matter, 39.
Microsporon furfur, 185.
Micturition, 247.
frequency of, 188.
pain after, 188.
pain before, 180.
Migraine, 239.
Milium, 179.
Mitral area, 84.
diastolic murmurs, 91.
disease, pallor in cases of, 17.
rapid pulse in, 96.
incompetence, 86–90.
cardiographic tracing in, 115.
causing thrill, 78.
sphygmographic tracing in, 113.
præsystolic murmurs, 91.
stenosis, 85, 87, 91.
cardiographic tracing in, 116.
causing thrill, 78.
overfilling of jugular veins in, 101.
systolic murmurs, 90.
valve, 83.
Molities ossium, bones in, 261.
propepton in urine of, 209.
Molluscum, 179.
Moore's test for sugar in urine, 214.
Motor functions of nervous system, 247.
of viscera, 247.
of voluntary muscles, 248.
Movements of eyeball, 240.
larynx, 132.
vocal cords, 132.
Mucous cloud in urine, 192.
Mucus in urine, 212.
Muguet, 28–32.
Murmurs, aortic, 93.
arterial, 99.
cephalic, 100.
endocardial, 87.
character of, 90.
point of maximum intensity of, 89.
propagation of, 89.
rhythm of, 89.
exocardial, 94.
hæmic, 94.
mitral, 90.
origin of, 87.
pulmonary, 93.
tricuspid, 92.
Muscles, contraction of, law of normal, 249.
contracture of, 261.
flaccidity of, 261.
functions of, 248.
ocular paralysis of, 241.
spasm of, 241.
paralysis of, 245.
reflex movements of, 251.
rigidity of, 261.
spasm of, 250.
tropho-neuroses of, 255.
twitching of, 251–260.
Muscular atrophy, progressive, 250.
tongue in, 29.
Muscular sense, 238.
Myelitis, electric conditions of, 249.
Myosis, 242.

N.

Nævi, 179.
Narcotic poisoning, 257.
Nares, examination of, 128.
inspection of, 129.
palpation of, 128.
Nasal speculum, 128.
Nausea, 38.

Nerve, abducens, paralysis of, 241.
inferior laryngeal, paralysis of, 133.
oculo-motor (3d), paralysis of, 240.
optic, 243.
recurrent laryngeal, paralysis of, 133.
superior laryngeal, paralysis of, 133.
trochlear or 4th, paralysis of, 241.
Nerves, ocular, paralysis of, 240.
spasm of, 241.
Nervous constitution, 22.
Nervous disease, excretion of phosphates in, 206.
Nervous system, 241–261.
cerebral and mental functions, 256.
condition of cranium and spine, 260.
motor functions, 247.
sensory functions, 233.
trophic functions, 255.
vaso-motor functions, 254.
Nœud vital, 248.
Neuralgia, 234.
intercostal, 119–234.
of chest wall simulating cardiac pain, 69.
salivation in, 31.
visceral, 234,
Neuritis, optic, 243.
Nitrate of silver causing argyria, 20
Nitric acid test for albumen in urine, 207.
Nitrite of amyl, inhalation of, 254.
Nodes on bones, 23, 261.
Normal, contraction, law of, 249.
temperature, 24.
Nostrils, *see* nares.
Notch, dicrotic, 105.
Numbness, 235.
Numeration of blood corpuscles, 66.
Nummular sputa, 124.
Nystagmus, 242.

O.

Obstruction, intestinal, indican excretion in, 202.
Ocular muscles, paralysis of, 241.
Oculo-motor nerve, 240.
Odor of urine, 192.
Œdema, causes of, 20.
glottidis, 132.
of lungs, percussion in, 154, 155.
sputum of, 158.
Œsophagus, auscultation of, 35.
examination of, 34.
obstruction of, 35.
causing retraction of abdomen, 44.
pain in, 35.
Omentum, tumors of, 45–51.
Ophthalmoscopic examination, 243.
Optic anæsthesia, 239.
disc, changes in, 243.
hyperæsthesia, 238.
hyperalgesia, 238.
nerve, condition of, 243.
neuritis, 243.
Organisms in blood, 65.
Os uteri, examination of, 232, 233.
Osteo-malacia, excretion of phosphates in, 206.
Ovarian disease, leading to amenorrhœa, 227.
tumors, diagnosis of, 229.
Ovaries, tumors of, 45–52.
Oxalate of lime in urine, 223.
Oxaluria, symptoms of, 224.
Oxyuris vermicularis, 42.

P.

Pain, 234.
cardiac, 70.
caused by stricture of urethra, 188.
by vesical calculus, 188.
girdle, 234.
in connection with respiratory system, 119.
in connection with urinary system, 188.
in loins, 188.
lightning, 235.
in supra-pubic region, 188.
of cystitis, 188.
of inter-costal neuralgia, 119.
of pleurisy, 119.
of pleurodynia, 119.
urethral, 188.
Palate, cicatrices in, after syphilis, 23.
form of, 33.
paralysis of, 33.

Pallor, causes of, 18.
Palpation of abdomen, 46–52.
abdominal aorta, 52.
arteries, 95.
circulatory organs, 76.
gall-bladder, 49.
intestines, 50.
kidneys, 52.
larynx, 129.
liver, 48.
mesenteric glands, 51.
nares, 128.
ovarian tumors, 52.
pancreas, 50.
respiratory system, 141.
stomach, 50.
Palpitation, 70.
Pancreas, palpation of, 50.
tumor of, 50.
Papules, 179.
Paræsthesiæ, 235.
Para-globulin in urine, 208.
Paralysis agitans, shaking of limbs, in, 251.
general, *see* general paralysis.
of crico-thyroid muscle, 133.
of inferior laryngeal nerve, 133.
of muscles of epiglottis, 133.
of ocular nerves, 240.
of superior laryngeal nerve, 133.
of tongue, 29.
of vocal cords, 129–132.
of voluntary muscles, 248.
varieties of, distinguished by electro-diagnosis, 249.
Paraplegia, 248.
vaso-motor affections in, 255.
Parasites, animal, of skin, 185.
vegetable, of skin, 182.
Parasitic affections of skin, 182.
worms in fæces, 42.
causing excessive appetite, 36.
Pareunia, 228.
Patellar reflex, 252.
Pectoriloquie aphonique, 175.
Pectoriloquy, footnote, 174.
Pediculus, 187.
Pepton in urine, clinical significance of, 211.
detection of, 211.
Percussion, feeling of resistance during, 160.
in ascites, 53.
Percussion, methods of, 152.
of abdomen, 53–60.
of heart, 79–82.
of kidneys, 58.
of liver, 54.
of lungs, 152.
of spine, 260.
of spleen, 57.
of stomach, 59.
regional, 162.
theory of, 147.
topographical, 161.
theory of, 155.
tympanitic, 156.
Percussion sound, intensity of, 149, 153.
pitch of, 150–155.
quality of, 151.
Pericardial effusion causing bulging, 137.
increase of cardiac dulness, 80.
friction, 82–94.
Pericardium, adherent, 72–73.
Perimeter, 239.
Perinæum, examination of, 232.
Peripheral paralysis, electric conditions of, 249–250.
Peristaltic movements, 46.
Peritoneal cavity, 47.
friction, 48.
Peritonitis, acute, breathing in, 138, 140.
drawing up of upper lip in, 21.
knees drawn up in, 23.
pain in, 47.
chronic, tenderness in, 47.
pelvic, leading to amenorrhœa, 227.
Perspiration, 20.
Per vaginam examination, 232.
Perverted sensations, 235.
Petechiæ, 181.
Phosphate of ammonium and magnesium in urine, 224.
of lime, amorphous, in urine, 224.
crystallized, 224.
of magnesium in urine, 225.
Phosphates in urine, 204.
quantity of, 206.
Phthiriasis, 180.
Phthisis, amenorrhæa in, 227.

Phthisis, blood in, 65.
blood pressure in cases of, 117.
bronchial breathing in, 168.
breathing in, 141.
causing shrinking of thorax, 137.
cough of, 120.
expiratory pressure in, 147.
form of thorax in, 136.
harsh breathing in, 165.
heredity of, 16.
jerky respiration in, 165.
moist râles in, 171.
pallor in cases of, 18.
paralysis of recurrent laryngeal nerve in, 133.
percussion in, 155, 158.
shrinking of apices of lung in, 162.
sputum of, 123, 124, 126, 127.
subclavian murmur in, 98.
sweating in, 20.
temperature, 24.
tubercule-bacillus in sputa of, 127.
vocal fremitus in, 142.
resonance in, 175.
Phymata, 179.
Pica, 36.
Pigeon-breast, 137.
Pigmentation of skin, 18, 19.
Pigments of urine, 190.
Pitch of percussion sound, 150–155.
Pitting of small-pox, 23.
Pityriasis versicolor, 180.
Plantar reflex, 252.
Plethora, complexion in, 18.
Pleuræ, thickening of, percussion in cases of, 154.
Pleural effusions, ægophony in, 175.
breathing in, 138–141.
causing enlargement of thorax, 137.
displacing apex beat, 76.
liver, 56.
modifying percussion note, 153, 158.
modifying vocal resonance, 175.
shrinking of thorax after absorption of, 138.
vocal fremitus in, 142.
Pleural friction, 143.
Pleurisy, breathing in, 138.
cough of, 120.
Pleurisy, friction sound, 173.
pain of, 119.
urea increased in, 199.
Pleuritic effusion, bronchial breathing above the level of a, 167.
friction, 173.
Pleurodynia distinguished from pleurisy, 119.
Plexímeter, vibrations of, 148.
Pneumatometer, 146.
Pneumonia, bronchial breathing in, 167.
causing enlargement of thorax, 138.
cough of, 120.
crepitant râle in, 170.
excretion of chlorides in, 203.
malar, flush of, 21.
percussion in, 154, 155, 158.
sputum of, 123, 124, 127.
temperature of, 24.
urea increased in, 198.
vocal fremitus in, 142.
resonance in, 175.
Pneumopericardium abolishing cardiac dulness, 81.
Pneumothorax, amphoric resonance in, 160.
amphoric respiration in, 169.
cracked-pot sound in, 159.
diminishing cardiac dulness, 81.
displacing apex beat, 76.
liver, 56.
metallic tinkling in, 172.
percussion note of, 158.
Poisoning, gastric pain after, 38.
Polarimeter, 216.
Poliomyelitis, anterior acuta, 250.
Pomphi, 179.
Posterior crico-arytenoid muscle, paralysis of, 134.
Præcordia, 71.
bulging of, 71.
depression of, 71.
Præsystolic mitral murmurs, 91.
tricuspid murmurs, 92.
Pregnancy, albuminuria in, 211.
bronzing of skin in, 19.
in its bearing on uterine disease, 227.
Pressure of air in respiration, 147.
points, motor, 251.
sensation of, 236.

Progressive muscular atrophy, 250.
tongue in, 29.
Propepton in urine, 209.
Prostate, hypertrophy of, frequency of micturition in, 188.
Prostatitis, pain of, 188.
Prurigo, 180.
Pruritus, 180–235.
Psoriasis, distribution of, 178.
fissures in, 180.
scales of, 180.
Pterygoid thorax, 137.
Ptosis, 240.
Pudenda, external, 230.
Pulex irritans, 187.
Pulmonary area, 84.
cavities, amphoric respiration over, 168.
bronchial breathing over, 168.
metallic tinkling in, 172.
sputum from, 123, 124.
vocal resonance over, 175.
consolidation, dry râles in, 173.
tympanitic note in, 159.
vocal resonance in, 175.
disease, cyanosis in, 18.
lips in, 26.
emphysema, 101.
murmurs, 93.
œdema, percussion in, 154, 155, 158.
sputum in, 122.
valves, 83.
Pulsation at root of neck, 73.
arterial, on thoracic wall, 75.
capillary, 75.
carotid, 72.
epigastric, 74.
in abdomen, 45.
jugular, 73.
of abdominal aorta, 52.
of cardiac apex, 72–76.
Pulse, radical, 95.
anacrotic, 106.
character of, 97.
dicrotic, 106–109.
expansion of, 97.
frequency of, 96.
hyperdicrotic, 110.
rhythm of, 96.
tension of, 97.
Pulse, volume of, 98.
Pulsus alternans, 97.
bigeminus, 97.
celer, 97.
intermittens, 97.
paradoxus, 96.
tardus, 97.
Pupil, Argyle-Robertson symptom, 243.
changes in the, 242.
contraction of, 242.
dilatation of, 243.
hippus, 243.
Purpura, 179.
menorrhagia in, 227.
Purpurin, 191.
Pus in urine, 220.
Pustules, 180.
Pyelitis, pain of, 188.
pus in urine in, 220.
Pylorus, incompetence of, 45.
Pyrexia, altering heart sounds, 85.
diminishing force of heart, 77.
dry skin of, 20.

Q.

Quality of percussion sound, 151.
Quantitative change of electric excitability, 249.

R.

Radial pulse, 95.
character of, 97.
expansion of, 97.
frequency of, 96.
rhythm of, 96.
tension of, 97.
tracings of, 105.
volume of, 97.
(*See* also Pulse.)
Râles, bubbling, 172.
crepitant, 170.
distinguished from friction, 174.
dry, 173.
moist, 171.
resonant, 172.
sibilant, 173.
sonorous, 173.
Rapidity of conduction of sensations, 237.
Reaction of degeneration, 250.
of urine, 194.

Recto-vaginal examination, 232.
Recurrent fever, 25.
paralysis, 133.
Redness of skin, 18.
Reduplication of heart-sounds, 86.
Reflex, abdominal, 252.
cremasteric, 248–252.
epigastric, 252.
gluteal, 252.
knee, 252.
plantar, 252.
scapular, 252.
Reflexes, deep, 252.
superficial, 252.
Regional percussion, 162.
Regions of thorax, 135.
Relapsing fever, 25.
Relaxation of lung tissue causing tympanitic note, 157.
cracked-pot sound in, 159.
Remittent fever, 24.
Renal calculus, pain caused by, 189.
dulness, 59.
tube-casts, 220.
Reproductive system, 226.
Resistance during percussion, 160.
Resonance, amphoric, 160.
explanation of, 150.
vocal, 174.
Respiration, amphoric, 168.
bronchial, 166.
broncho-vesicular, 169.
Cheyne-stokes, 139.
extent of movements, 141.
frequency of, 138.
nervous mechanism, 248.
rhythm of, 139.
type of, 140.
vesicular, 164.
Respiratory centre, disease of, 139.
movements of thorax, 138.
râles, 170.
system, 119.
auscultation, 163.
examination of nares and larynx, 128.
inspection, 135.
laryngoscopic examination, 130.
mensuration, 143.
pain in connection with, 119.
palpation, 141.
Respiratory centre, percussion, theory of, 147.
subjective phenomena, 119.
Retina, appearance of, 244.
Retinal vessels, 243.
Retro-pharyngeal abscess, 33.
Rhagades, 180.
Rheumatic constitution, 22.
paralysis, electric conditions of, 249.
Rheumatism, acute, acid urine in, 195.
joint affections in, 261.
sweating in, 20.
urea increased in, 199.
uric acid excretion in, 200.
Rhonchus, 171, footnote.
Rhubarb, urine colored by, 191.
Rhinoscopy, 129.
Rickets, bones in, 261.
form of thorax in, 137.
large joints in cases of, 23.
Rigidity of muscles, 261.
Ringworm, 183.
Risus sardonicus, 27.
Rupia, 180.

S.

Saccharimeter, 215.
Saliva, 31.
diminution of, 31.
increase of, 31.
reaction of, 31.
Salivation, 31.
Sanguine constitution, 21.
Santonin, urine colored by, 191.
Sarcinæ ventriculi, 39.
Sarcoptes scabiei, 185.
Saturnine paralysis, 250.
Scabies, 180–185.
Scales, 180.
Scapular reflex, 252.
Scarlet fever, desqumation of, 180.
throat in, 32.
tongue in, 30.
Scars, 180.
Sciatica, 234.
Scleroderma, fissures of, 181.
Scorbutus, *see* scurvy.
Scotoma, 239.
Scurvy, spongy gums in, 28.
Second sound of heart, 86.

Sediments, urinary, 219.
of acid urine, 222.
of alkaline urine, 224.
table of inorganic, 222.
Semi-circular canals, disease of, 253.
Senna, urine colored by, 191.
Sensation, common or general, 236.
of pressure, 236.
of locality, 237.
of temperature. 237.
tactile, 236.
Sensations, perverted, 235.
subjective, 234.
Sense of hearing, 244.
of sight, 238.
of smell, 246.
of taste, 245.
Sensibility, common cutaneous, 236.
Sensory functions, nervous system, 233.
Serum-globulin in urine, 209.
clinical significance of, 211.
Sexual function, 248.
Shrinking of thorax, 137–144.
Sight, sense of, 238.
Sixth nerve, paralysis of, 241.
spasm of, 242.
Skin, blueness of, 18.
bronzing of, 19.
elements of, involved in the eruption, 178.
eruptions, 177.
grayness of, 20.
pitting of, 20.
trophic affections of, 256.
Skull, condition of, 260.
Sleep, 259.
Small-pox, eruption of, 180.
pitting of skin after, 23.
swelling of tongue in, 29.
temperature of, 24.
Smell, sense of, 246.
Solids of urine, 193.
Somnambulism, 259.
Somnolence, 259.
Sordes on lips, 26.
Sore throat, 32.
Sound, uterine, 264.
Sounds, adventitious, accompanying respiration, 170.
in arteries, 98.
Sounds of the heart, 83.
enfeeblement of, 86.
impurity of, 86.
intensification of, 86.
modifications of, 85.
reduplication of, 86.
Spasm of ocular nerves, 241.
of sixth nerve, 242.
of third nerve, 241.
of voluntary muscles, 250.
Specific gravity of urine, 192.
Speculum, nasal, 128.
vaginal, 233.
Speech, affections of, 258.
Spermatozoa in urine, 221.
Sphygmograph, 104, 111.
Sphygmographic tracing, healthy, 105.
Sphygmomanometer, 116.
Spinal cord, cilio-spinal region, 242.
paralysis, electricity in, 249.
Spine, condition of, 260.
curvature of, 260.
percussion of, 260.
Spirochæte Obermeierii, 66.
Spirometer, 145.
Spleen, consistence of, 50.
displacements of, 58.
enlargement of, 50–58.
palpation of, 49.
percussion of, 57.
position of in abdomen, 57.
tumors of, 45.
Splenic fever, 65.
Sponge tents, 234.
test, hot, 260.
Spongy gums in scurvy, 28.
Sputa, 121.
bronchial casts in, 126.
Charcot's crystals in, 127.
chemical characters of, 122.
epithelial cells in, 125.
lung tissue in, 126.
micro-organisms in, 127.
microscopic examination of, 124.
physical characters of, 123.
tubercule-bacillus in, 127.
varieties of, 122.
Squamæ, 180.
Squint, 240.
Stellar phosphate in urine, 224.
Stenosis of larynx, 146, 147.

Stenosis, of trachea, 146, 147.
Stethograph, 145.
Stethoscope, 83, 163.
Stomach, examination of, 44.
palpation of, 50.
percussion of, 59.
position of, 59.
tumor of, 45, 51.
Stomatitis, 28.
Stone in bladder, pain caused by, 188.
Strabismus, 240.
"Strawberry tongue," 30.
Strychnine-poisoning, risus sardonicus in, 27.
Strumous constitution, 22.
Subcutaneous emphysema, 20.
œdema, 20.
Subjective sensations, 234.
Succussion, hippocratic, 176.
Sugar in urine, estimation by Pavy's method, 214.
by circular polarization, 215.
tests for, 214.
Sulphates in urine, detection and estimation of, 203.
quantity excreted, 204.
Sulphocyanic acid in urine, 204.
Sulphur in urine, 205.
Sulphuretted hydrogen in urine, 205.
Superficial reflexes, 252.
Superior laryngeal nerve, paralysis of, 133.
Supra-renal capsules, disease of, causing bronzing, 19.
Swallowing, 247.
Sweat, critical, 20.
Swelling of ankles, 20.
Sycosis menti, 180, 183.
Sympathetic resonance, theory of, 150.
Syncope, 70.
Syphilides, 178, 179.
Syphilis, 62.
crusts, 180.
fissures of, 26, 180.
hereditary, 16.
ocular paralysis in, 241.
pallor of, 23.
signs of former, 23.
teeth in, 27.
tongue in, 30.
Systolic heart-sound, 83, 84.
Systolic, indrawing of thoracic wall, 72.
murmur, aortic, 93.
mitral, 90.
pulmonary, 93.
tricuspid, 92.
vesicular breathing, 165.

T.

Taches cérébrales, 254.
Tactile sensation, 236.
Tænia mediocanellata, 42.
solium 42.
Tangle tents, 235.
Tape-measure, 143.
Taste, sense of, 245.
Taurin in urine, 204.
Teeth, 27.
Temperament, 21.
Temperature, 23.
sensibility to, 237.
Tenesmus, 40.
Tension of chest wall influencing percussion, 155.
pulmonic tissue influencing percussion, 155.
Tents, 235.
Test types, 239.
Tests for albumen in urine, 207.
Tetanus, risus sardonicus in, 27.
Theory of percussion, 147.
Thermo-anæsthesia, 237.
-hyperæsthesia, 237.
Thermometer, 23.
Third nerve, paralysis of, 240.
spasm of, 241.
Thirst, 36.
Thoracic duct, 61.
Thoracic wall, modifying percussion, 154.
fluctuation in, 143.
tension of, influencing percussion, 155.
Thoracometer, 145.
Thorax, antero-posterior diameter, 144.
Thorax, circumference of, 143, 144.
enlargement of, 137, 144.
form of, 137, 141.
heteromorphisms of, 137.
measurement of movements of, 145.
movements of, 141.

Thorax, regions of, 135.
respiratory movements of, 138.
shrinking of, 137, 144.
vibrations of wall of, 148.
Throat, sore, 32.
Thrombosis, 101.
Thrills, endocardial, 78.
Thrush, 28, 31.
Thymus gland, 64.
Thyroid gland, 63.
Tic douloureux, 234.
Tight-lacing, effect of, on liver, 49.
Tinea circinata, 183.
favosa, 182.
sycosis, 180, 183.
tonsurans, 183.
Tinkling, metallic, 172.
Tongue, 28.
form of, 28.
fur on, 30.
movements of, 29.
surface of, 29.
Tonic spasm of voluntary muscles, 250.
Tonsillitis, 32.
Topographical percussion, 161.
theory of, 156.
Tracings, cardiographic, in health, 112.
measurement of, note on, 117.
sphygmographic, in health, 105.
Tracheal note of Williams, 159.
Transparency of urine, 192.
Traumatic paralysis, electric conditions of, 249.
Tremor, 251.
Trichocephalus dispar, 207.
Trichophyton, 183.
Tricuspid area, 84.
incompetence, 92.
jugular pulsation in, 73.
præsystolic murmurs, 92.
stenosis, 92.
systolic murmurs, 93.
valve, 83.
Triple phosphate in urine, 224.
Trochlearis, nervus, paralysis of, 241.
Trophic functions, 255.
Tube-casts in urine, 220.
epithelial, 220.
fatty, 220.
granular, 221.
Tube-casts in hyaline, 221.
pus, 221.
waxy, 252.
Tubercular meningitis causing retraction of abdomen, 44.
peritonitis causing ascites, 54.
Tubercule-bacillus in sputa, 127.
Tubercules, 179.
Tubular respiratory murmur, *see* bronchial.
Tumefaction of larynx, 132.
Tumors, cerebral, ophthalmoscopic appearances in, 244.
fæcal, 51.
of abdomen, 45.
of kidneys, 45, 52.
of liver, 45, 48.
of mesenteric glands, 51.
of omentum, 45, 51.
of ovaries, 45, 52.
of pancreas, 50.
of skin, 179.
of spleen, 45, 49.
of stomach, 44, 50.
Tuning-fork test, 244.
Tupelo tents, 235.
Twitching, muscular, 251, 261.
Tympanitic percussion, 156.
Types, test, 239.
Typhoid state, expression in, 21.
Typhus fever, temperature of, 25.
urea increased in, 198.
Tyrosin in urine, 224.

U.

Ulceration of larynx, 132.
Ulcers, 181.
Umbilicus, distension of veins at, 44.
Uræmia, convulsions of, 251.
diarrhœa caused by, 40.
respiration in, 140.
urea in perspiration of, 46.
Urate of ammonium in urine, 226.
Urates as an amorphous deposit in urine, 222.
Urea, estimation of, 195.
quantity excreted, 199.
in perspiration, 20.
Urethritis, pain of, 188.
pus in urine in, 219.
Uric acid as a urinary deposit, 223.
detection of, 199.

Uric acid, estimation of, 199.
in urine, 199.
quantity excreted in health, 199.
Urinary system, 188–226.
Urine, abnormal constituents of, 206.
acid, sediments found in, 222.
acidity of, 194.
albumen in, 206.
alkaline, sediments of, 224.
alkalinity of, 194.
ammoniacal, 194.
ammonio-magnesian phosphate in, 224.
amorphous phosphate of lime in, 224.
bile acids in, 218.
pigment in, 218.
blood in, 217.
corpuscles in, 219.
carbonate of lime in, 226.
chlorides in, 202.
cholesterin as a deposit in, 224.
chylous, 62.
color of, 189.
creatinin in, 200.
crystallized phosphate of lime in, 224.
cystin as a deposit in, 224.
epithelium in, 220.
examination of, 189.
fermentation of, 194.
fibrin in, 210.
indican in, 201.
leucin and tyrosin as deposits in, 224.
melanin in, 191.
micro-organisms in, 221.
mucous cloud in, 192.
mucus in, 212.
normal constituents of, 195.
organic deposits, 219.
oxalate of lime as a deposit in, 223.
para-globulin in, 209.
pepton in, 211.
phosphate of magnesium in, 225.
phosphates in, 204.
pigments of, 190.
propepton in, 209.
purpurin in, 191.
pus corpuscles in, 219.
Urine—*continued.*
quantity of, 189.
reaction of, 194.
serum-globulin in, 209.
solids of, 193.
specific gravity of, 192.
spermatozoa in, 221.
stellar phosphate in, 224.
sugar in, 212.
sulphates in, 203.
transparency of, 192.
triple-phosphate in, 224.
tube casts in, 220.
urate of ammonium in, 225.
urates as a deposit in, 223.
urea in, 195.
uric acid as a deposit in, 223.
uric acid in, 199.
uro-bilin in, 190.
uro-erythrin in, 191.
Urinometer, 192.
Uro-bilin, 190.
Uro-erythrin, 191, 222.
Uroxanthin in urine, 201.
Urticaria, 179.
Uterine canal, occlusion of, leading to amenorrhœa, 227.
disease, causing vomiting, 38.
sound, 234.
tents, 235.
Uterus, dilatation of canal of, 235.
diseases of, leading to menorrhagia, 227.
to dysmenorrhœa, 228.
displacements of, 235.
rudimentary, leading to amenorrhœa, 227.

V.

Vagina, occlusion of, leading to amenorrhœa, 227.
Vaginal examination, 232.
speculum, 233.
Valves of the heart, 83.
Vaso-motor affections, cutaneous, 254.
visceral, 255.
functions, 254.
Veins, 100.
auscultation of, 101.
bruit de diable in, 101.
cervical, 101.
inspection, 100.

Veins, overfilling of, 101.
pulsation of, 72.
undulation of, 101.
Vegetable parasites of skin, 182.
Venous humming murmur, 101.
pulse, 70.
Vertigo, 235.
labyrinthine, 253.
Vesical calculus, pain caused by, 188.
Vesicles in skin, 179.
Vesicular breathing, 164.
Vessels, lymphatic, 61.
Vibrations of air in lungs, 148.
of pleximeter, 148.
of thoracic wall, 148.
Visceral motor functions, 247.
neuralgia, 234.
sensations, 236.
trophic affections, 256.
vaso-motor affections, 255.
Vision, alterations in extent of field of, 239.
alterations in perception of colors, 240.
diminution of acuteness of, 239.
Vital capacity, 145.
Vocal cords, cadaveric position of, 133.
paralysis of, 129, 132.
Vocal cords, fremitus, 142.
resonance, 174.
Voice, auscultation of, 174.
huskiness of, in laryngeal affections, 129, 133.
Voluntary muscles, functions of, 248.
reflex movements of, 251.
spasm of, 250.
Vomicæ, pulmonary, amphoric resonance in, 160.
amphoric respiration over, 168.
bronchial breathing over, 168.
cracked-pot sound in, 159.
metallic tinkling in, 172.
percussion of, 158.
vocal resonance over, 175.
Vomited matter, 39.
Vomiting, causes of, 38.
Vulva, examination of, 229.

W.

Water-brash, 38.
Weight, 17.
Wheals, 179.
Whooping-cough, 121.
Williams' tracheal note, 159.

Y.

Yellow fever, vomiting of blood in, 39.

DUE DATE

AUG 7 - 1991

AUG 2 8 1991

AUG 8 - 1991

Printed
in USA

COLUMBIA UNIVERSITY LIBRARIES
0023788917

DUPLICATE
HX00014338